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DISEASES OF
THE DIGESTIVE SYSTEM

MODERN CLINICAL MEDICINE

DISEASES
OF THE
IGESTIVE SYSTEM

EDITED BY

FRANK BILLINGS, M.D.

PROFESSOR OF MEDICINE, UNIVERSITY OF CHICAGO, AND PROFESSOR OF
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AN AUTHORIZED TRANSLATION FROM "DIE DEUTSCHE KLINIK"
UNDER THE GENERAL EDITORIAL SUPERVISION OF
JULIUS L. SALINGER, M.D.

WITH FORTY-FIVE ILLUSTRATIONS IN THE TEXT

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INTRODUCTION

THE advances of chemistry in the last few decades have placed the diagnosis and treatment of Digestive Diseases upon a firm foundation. Much that was formerly purely theoretical and based upon hypothesis has now become almost absolute knowledge. The study of metabolism has lifted the veil of mystery from much that was obscure. In the investigation of diseases of digestion, in which physiology, chemistry, and pathology are so intimately connected, the practitioner has received valuable aid in relieving suffering humanity. In practice, perhaps the greatest benefits may be conveyed by the correction of even the most insignificant alimentary errors.

In no organ of the abdominal cavity, and perhaps of the entire body, are the functions so manifold and diverse as in the pancreas. Text-books dating back a few decades scarcely mentioned the name of the organ, not to speak of a reference to its diseases. Thanks to the labors of investigators quite a literature is to-day extant, and the search-light of diagnosis is beginning to be thrown upon maladies of this important digestive organ.

This volume treats not only of these subjects, but a new study has been opened, or, more properly, revived; namely, the investigation of the feces. What the examination of the sputum is to disease of the lungs, what the examination of the urine is to disease of the kidney and to disorders of metabolism, what the analysis of the gastric contents is to the diagnosis of disease of the stomach, the macroscopic, microscopic, bacteriologic and chemical investigation of the feces is to disease of the intestines.

Abdominal surgery has aided much in the elucidation of many of these problems, and although we have reached but the first step of the ladder in the ascent of the tree of knowledge, much has been learned and still more may be expected.

38900

EDITOR'S PREFACE

TO-DAY diseases of the Digestive Tract stand in the forefront of subjects which interest the practitioner and the surgeon.

Many of the diseases included in this volume lie on the borderland of medicine and surgery.

This volume includes articles from many of the most eminent men of Europe, specialists in internal medicine and in diseases of the digestive tract.

The subjects are treated very fully and at the same time in a concise and practical manner. The modern methods of examination, including physical and chemical measures, are clearly set forth, which will enable the practitioner to apply them with the same ease that he may make a physical examination of the chest and a chemical and microscopic uranalysis.

The diagnosis of the various diseases is fully discussed and the treatment, including the dietary, is satisfactorily full and complete. Indeed, the subjects are so fully treated that the editor found it inexpedient to add to or to modify the text to any important extent.

The translator has done the work well, with the result that the text is smooth and interestingly readable.

FRANK BILLINGS.

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LIST OF CONTRIBUTIONS

Stenosis of the Esophagus. By Th. Rosenheim, Berlin.

The History and Clinical Indications of Gastric Lavage. By W. Fleiner, Heidelberg.

Functional Diseases of the Stomach. By H. Leo, Bonn.

The Diagnostic and Therapeutic Significance of Secretory Disturbances of the Stomach. By H. Strauss, Berlin.

Diagnosis and Treatment of Gastric Dilatation. By F. Riegel, Giessen.

Gastric Ulcer and Gastric Hemorrhage. By C. A. Ewald, Berlin.

Gastric and Intestinal Carcinomata. By J. Boas, Berlin.

Displacements of the Abdominal Viscera and of the Heart. By F. Hirschfeld, Berlin.

Symptomatology of the Diseases of the Pancreas. By L. Oser, Vienna.

Jaundice and Hepatic Insufficiency. By O. Minkowski, Cologne.

Chronic Inflammation of the Liver. By E. Stadelmann, Berlin.

Neoplasms of the Liver and Biliary Passages. By Fr. Kraus, Graz.

Echinococcus of the Liver. By E. Stadelmann, Berlin.

Gall-stones. By E. Neusser, Vienna.

Acute Diffuse Peritonitis, Appendicitis, and Perityphlitis:

- I. Diffuse and Circumscribed Peritonitis. By O. Vierordt, Heidelberg.
- II. Chronic Perityphlitis. By J. Boas, Berlin.

Examination of the Feces. By J. Strasburger, Bonn.

Diarrhea, Intestinal Catarrh, and Intestinal Tuberculosis. By W. Fleiner, Heidelberg.

Constipation and Hemorrhoids. By J. Boas, Berlin.

Mucous Colic and Membranous Intestinal Catarrh. By G. Hoppe-Seyler, Kiel.

Intestinal Constriction and Intestinal Occlusion. By H. Nothnagel, Vienna.

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DISEASES OF THE ESOPHAGUS AND OF THE STOMACH

2

STENOSIS OF THE ESOPHAGUS

By TH. ROSENHEIM, Berlin

ETIOLOGY

Diseases of the esophagus are, as a rule, infrequent. Almost invariably, however, they are of great importance, as an essential function of the organ is rapidly influenced, and because these processes occur in the immediate vicinity of the most vital organs, so that a dangerous transmission of the disease may readily take place. As the organ in question is one of the least sensitive ones, pain in disease of the esophagus is rare. In the main, there is but one symptom: *disturbance of deglutition.* This occurs in varying degrees of intensity in *all* esophageal affections, yet the severity of the causal process has no decisive influence upon the degree; for the hindrance in deglutition may be slight in carcinoma, and, on the contrary, it may be high-graded in small erosions, or in derangement of innervation. The basis for most disturbances of deglutition is a *stenosis of the lumen,* which may arise from manifold causes, and to which this article will be exclusively devoted. The majority of diseases occurring in the esophagus must be more or less considered; this will broaden our theme to a great extent, but we shall confine ourselves well within the required limits if we emphasize only the most important features from a *diagnostic* and *therapeutic standpoint.* The researches of the last few years, and especially since the introduction of esophagoscopy, have revealed much that is new and important.

The most frequent and, practically, the most significant cause of stenosis of the esophagus is *cancer;* other diseases are rarely mentioned, at most, here and there, stricture due to swallowing of caustics. But the forms of stenosis are by no means exhausted with this, and the spastic stenoses, particularly, call for more thorough consideration than was formerly devoted to them. *Disturbances in innervation play an important part in the origin of derangement of deglutition.* Yet it is often extraordinarily difficult accurately to measure the part which they play in the causation of the symptoms of the disease, as they rarely occur alone, but usually are combined with other pathologico-anatomical changes in the esophagus or adjacent organs. We must, however, always endeavor to analyze as accu-

dysphagia, which is the usual course, we observe in some cases in the midst of the best of health and without prodromes a sudden and decided impediment to deglutition. At other times, decided variation in the degree of passability of the esophagus may be noted. On some days the patient can apparently swallow anything, on other days there is an obstacle. Doubtless the condition of the nervous system here plays an important rôle, for I have repeatedly observed this in very nervous patients with carcinoma of the esophagus. With a prolonged course of the affection, amelioration may come by the purulent softening and ichorous decomposition of prominent cancer proliferations; the patient again becomes hopeful, but this improvement is only the precursor of the final catastrophe.

The following fact is also remarkable: The patients complain of increasing difficulty in deglutition, so that even fluids fail to pass, or only with great effort, but, in this condition it is always possible to pass esophageal bougies into the stomach. *This disproportion between the natural power of deglutition and the results of probing* is found in all the varied affections of the esophagus, but chiefly in *compression of the organ* and in *neuroses;* occasionally, however, it is also observed in flat carcinomata situated in the wall of the organ and not distributed in annular form, since these offer no marked resistance to the sound, which readily passes them, and they disturb the active deglutition only in a reflex manner. Thus *spasms* are caused especially at the upper portion of the diseased area, but they may also occur in normal portions of the canal. For example, as not infrequent complications, we may have spasms behind the larynx, provided the tumor is situated in the cardia, and, in addition to this, very disagreeable *sensations of constriction* are felt at the boundary line between the pharynx and esophagus, and these necessitate continued efforts at swallowing on the part of the patient; this forms a very usual complication in the various diseases of the esophagus, and particularly in carcinoma, immaterial what its situation. The reports of the patient as to the region in which he feels constriction and pressure are often inaccurate and misleading. Frequently he will state that the bolus is arrested in the neck, while the cancer is situated in the cardia, or vice versa; the distress is sometimes localized in the epigastrium, when the neoplasm has developed in the upper half of the esophagus. *Therefore, the origin of disturbances of deglutition is the combination of a series of factors.* At one time, *mechanical obstruction* such as is presented by the infiltration of the wall of the esophagus plays an important rôle; at another, we observe *increased sensitiveness of the nerves of the muscular apparatus* which, as soon as the food reaches the inflamed area, readily show abnormal reaction. This reaction is the result of irritation, and is usually manifest as a spasm at the seat of the affection or in distant areas. Added to this are disturbances due to *inflammatory processes* in the mucous membrane of the areas surrounding the cancer, alone or in combination with an *atonic-ectatic condition* of the organ

rately as possible the genesis of the difficulty in swall
decisive importance in the therapy.

In reviewing the morbid processes in the esophag
question under the picture of stenosis, the enormous
may be divided into two groups: In the first group
abnormal processes *which have their seat in the organ i*
directly from the organ, and thus narrow its lumen; i
we consider the cases in which stenosis is due to *comp*
be mentioned tumors of the mediastinum, of the vert
or enlarged lymph-glands, aortic aneurysm, peri-esoph
finally, filled diverticula which produce stenosis by pres
To the first group belong: (a) neoplastic stenoses
and cicatricial stenoses; (c) spastic stenoses; (d) here
lastly, (e) the obstruction to the propulsion of the in
produced by the esophagus, in which sense foreign bo
swallowed, polypi with long pedicles, and aphthous sto

SYMPTOMS

Let us now describe the *subjective and objective sy*
of the esophagus in the various forms of the disease u
considered, and let us observe the differences in the
sented by the several types. For this purpose it is
symptoms of cancer of the esophagus, the affection t
causes stenosis of the esophagus. How frequent carcin
nitely seen from statistics which show that about 60 per
of the esophagus are of a malignant nature, accordin
tions even 90 per cent.; but from my own experience
figure somewhat too high. It may be remarked in
of the esophagus occurs in different countries with varia
example, in Moscow unquestionably the cases are more
Berlin.

Difficulty or an impediment in deglutition, therefor
most constant symptom of cancer of the esophagus. It
also, which makes the patient anxious, and leads him to
The disturbance in swallowing appears gradually as m
times behind the corpus sterni and behind the larynx,
of the stomach and noted upon swallowing solid food;
even if well masticated, pass only with the greatest diffi
taneously with fluid; often they are arrested; then com
watery, pappy food can be forced into the stomach only
be swallowed in small portions, slowly and carefully;
is entirely occluded, transitorily or permanently, so tha
starvation. Besides this gradual, insidious, progressi

dysphagia, which is the usual course, we observe in some cases in the midst of the best of health and without prodromes a sudden and decided impediment to deglutition. At other times, decided variation in the degree of passability of the esophagus may be noted. On some days the patient can apparently swallow anything, on other days there is an obstacle. Doubtless the condition of the nervous system here plays an important rôle, for I have repeatedly observed this in very nervous patients with carcinoma of the esophagus. With a prolonged course of the affection, amelioration may come by the purulent softening and ichorous decomposition of prominent cancer proliferations; the patient again becomes hopeful, but this improvement is only the precursor of the final catastrophe.

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above the neoplasm. In regard to the last point, it m
that *hypertrophy,* and also *dilatation,* may develop abov
that the latter, however, only rarely is decided, as a pr(
nent stagnation of food does not readily take place. On
the bolus is arrested, it is usually soon regurgitated.
naturally occurs the more promptly the greater the dis
the narrowness of the stricture and the consistency o
as between the local sensitiveness and irritability of th
regurgitation occurs immediately after swallowing, or f
to a quarter of an hour after, rarely later. When delay
tion is usually present. In this case, if the esophageal so
of food may be brought up in which the absence of p
clearly that they have never reached the stomach.
processes of decomposition must arise when the food
time in the esophagus; consequently the patients not ra
fetid odor from the mouth. But these processes may oc
stage of the disease even without a decided retention of
decomposition of the cancer, and thus give rise to offen

Besides food, and even independently of its intr(
mucoid fluid having a fetid odor, is often ejected, an(
traces of blood. This is the product of the inflamma
accompanies cancer of the esophagus, and which some
to the pharynx and larynx. The combined effect of the
is *cough,* which often increases to extremely severe paro
ally distress the patient; added to this is the fact that br
culous changes in the lungs commonly coexist with cance

Apart from the unpleasant sensation of pressure m
by the arrest of the *bolus, the affection may be painle*
or even permanently, for the esophagus is one of the lea
and in this malady only an implication of the medias
and the pericardium, the compression of important ner
vicinity, the implication of the vertebral column and th
be productive of pain. Then *boring, burning, lancinating*
accompany the act of deglutition or follow it, but may
ently of the introduction of food, particularly at nigh
felt in the intercostal spaces, between the shoulder bla(
trium, in the throat and head; for example, frequently in
extremities. It is obvious that by the metastatic distrib
further growth of the cancer, manifold symptoms ma
shall here mention, for example, *vasomotor and trophic*
nails; also the occurrence of *inequality of the pupils;* alm
pupil is contracted. *Paralysis of the recurrent laryngea*
deserves special mention. Unilateral paralysis of the vo
common symptom. As this may not cause any change i

it can be detected only by a laryngeal examination, and thus is frequently overlooked. Often, however, it is an early symptom, and may even be present when as yet the stenosis is scarcely obvious.

Dyspnea and attacks of pain resembling angina pectoris arise in the course of the disease either because the neoplasm posteriorly presses immediately upon the trachea, or upon both recurrent laryngeal nerves, or implicates the posterior plate of cricoid cartilage and its surroundings, in which case the nerve and the muscular substance of the dilators of the vocal cords are directly destroyed.

Perforation of the esophageal wall may take place without producing special symptoms, and this is true even if the formation of a *fistula between the esophagus and the trachea or the bronchi* has already occurred, provided that the opening is small and the esophageal stricture slight, so that food may pass the organ without being arrested; otherwise, severe *paroxysms of cough and marked dyspnea upon the ingestion of food* are the characteristic signs of this last-named complication. The paroxysms of cough cause the ejection of mucus stained with blood simultaneously with particles of food which have found their way into the respiratory apparatus; sooner or later, upon continued nutrition by the mouth, pneumonia or pulmonary gangrene occurs. If the fistulous passage is very narrow, and the stricture still relatively permeable, it is sometimes noted that thin fluids cause paroxysms of cough, while more compact food passes with ease.

Decided spontaneous *hemorrhages* from cancerous ulcers in the esophagus which cause hematemesis are rare symptoms of carcinoma. More frequent are fatal *internal* hemorrhages due to the *erosion of a vessel* with the advance of the process.

Concerning the difficulties on the part of the *gastrointestinal canal* and the general condition: At the onset of the affection the patients usually have a good *appetite*, and in some pitiable cases this continues until death. In the further course of the disease, as a rule, hunger becomes less pressing, while burning *thirst* almost invariably persists. Stubborn *constipation* is common. In advanced cases, after the food has reached the stomach, it sometimes produces a continuous *dull pain in the epigastrium*, not rarely accompanied by eructations and nausea. These dyspeptic symptoms have no direct significance in cancer of the esophagus. They are to be attributed to secondary inflammatory atrophic processes in the mucous membrane of the stomach which are sooner or later added, and the more rapidly, the closer the cancer lies to the stomach, and the sooner it implicates this organ.

It is obvious that *inanition and cachexia* will early develop in the clinical picture, according to the nature of the disturbances which the affection produces. At first the loss in strength is parallel with the duration of the disease and the degree of obstruction to deglutition. If it is possible to diminish, or even to remove, the latter, although only temporarily, the patients, as a rule, rapidly recover and in Exceptionally carcinoma of the esophagus, without any f ance, causes extreme debility by a deleterious and toxi proteid metabolism.

Examination of the patient in the various stages of nishes results which are further modified according to th culty and the complications. *Inspection and palpation* us to recognize *enlargement of the lymph-glands* in the su cervical regions; but the utilization of this finding ne since quite marked enlargement of the glands is also f tuberculous individuals, a moderate enlargement not infr lis and catarrhal affections of the organs of the throat *present without a recognizable disease*. If the cancer i upper portion of the esophagus it may be externally pal beside the trachea and the larynx, and if it attack the cr pharynx it may even be *seen* by the laryngoscope.

When the cancer is deeply located, and cannot be th must *investigate by means of the sound*. We should fir *no disease is present which contraindicates the use of the* attempt to pass the stricture with a thick instrument (diameter). After it is within the esophagus it should be *fully* forced; if resistance is met with, it must be overcome but boring should be avoided; upon withdrawal *the leng portion from the teeth should be carefully noted.* It will pen that the figures gained in this manner will vary se upon different days—this is explained by the fact that to a different depth, or the point is embedded in the strict in spite of these variations, they enable us to locate the give us no idea of its extent. The resistance that the s be in the upper, in the middle, or in the lower part o neoplasm. It is a valuable diagnostic aid *carefully to exa of the sound*. In this we frequently find remains of mucus, and sometimes even *shreds of tissue* which unde permit the exact recognition of the disease. If it be im nize the characteristic cancer cell nests in the investigat preparation, then the suspicious portion may be examine preparation of sections after hardening in alcohol, or, a m the frozen section with the microtome and its staining (f picro-carmin).

After the presence of a stenosis has been determined thick sound, we should attempt to *pass through the narr more slender instruments;* frequently it will be impossi stomach with the finest instruments, although pappy and, o solid food still pass the stricture. The explanation of th the point of the sound is caught in a diverticulum above the narrowed area, or the canal of the stricture is not straight but follows an irregular, spiral course. On the other hand, the patients may complain of difficulty in swallowing food in a certain area of the esophagus, yet the largest sound finds no resistance in its entire course to the stomach; *in cases of beginning cancer formation, particularly, the results of examination may be negative.* Here the latter course must explain the case, or, if an *early diagnosis* is desired, we must investigate by means of *the esophagoscope.* The narrowed area which has been revealed by the sound, even in advanced cases, may be entirely unrecognizable if extensive ulceration and rapid destruction have occurred; but here also the esophagoscope gives the desired information.

The esophagoscopic pictures show great variations, which certainly depend upon *the stage* of development of the neoplasm, and, above all, *the nature of the growth.* The picture which presents itself is entirely different when *cauliflower-like proliferations* enter into the lumen, or, on the contrary, when only *an induration* of the wall has occurred, when *ulcer formation* has taken place, when *the mucous membrane is still retained,* when *the narrowing* is very decided, or when it is very slight.

The cancerous protuberances which narrow the space, if accurately investigated, cannot be confounded with anything else; there is no process in the esophagus except carcinoma that runs its course with the formation of these *prominent, whitish-grey to greyish or dirty greyish-yellow nodules permeated by punctiform hemorrhages.* The finding of a prominence covered with mucous membrane, however, may be of varying significance.

Even in advanced cases of carcinoma the stenosis is generally not due to cancerous masses which have proliferated into the lumen, but is due to *infiltration,* and this, viewed from above, presents itself as a swelling or a round tumor about the size of a cherry with *well-retained mucous membrane,* while ulceration or splitting of the surface cannot be detected because it occurs only toward the inner wall, therefore, arises in the lumen of the stricture. Frequently, above these protuberances the mucous membrane appears paler than normal, sometimes more yellow or, cyanotically, bluish-red, occasionally even loosened and markedly reddened. Worthy of note are the *epithelial thickenings* in different forms (streaks, flakes, etc.), which resemble leukoplakia of the tongue, and are found in the immediate surroundings of the morbid focus. At other times *whitish, papillary excrescences,* resembling pointed condylomata, or papillæ consisting of several whitish papillary proliferations, are met with in front of the narrowed area, frequently situated upon a perfectly normal mucous membrane. These last mentioned changes are *not pathognomonic of carcinoma;* we also meet with them in other conditions.

Not rarely the wall is deeply infiltrated only in the cancerous mass; then the formation of the above-mentioned one-sided protuberance does not occur, but frequently there is an *annular* narrowing constriction downward. We can then pass the tube as area, but no farther, and we descry from above a funn with mucous membrane folds converging downward. Sc trance into the stricture may be distinctly perceived, eve area, occasionally, however, the folds so overlap that this is

A similar picture is sometimes presented to us by caustics, and the previously mentioned protuberances w mucous membrane may also be produced by the bulging c esophagus in consequence of compression from without. *proof of ulceration* or of a *cauliflower-like proliferation* lumen is of the greatest importance. Sometimes it is pos the line of demarcation where the healthy mucous mem having an eroded appearance, or the transition to the ul surface of which can no longer be recognized, betrays itse by free, floating shreds of mucous membrane. We ma ulcerated portion by the *dirty, greyish-yellow, purulent coc* and beside which the *greyish-red, uneven cancer surface* ca be brought to view by cleansing, and which is characteriz *to continuous capillary hemorrhages.* Sometimes after th the tube, blood oozes continuously from the ulcerated sur of careful cleansing with cotton, nothing more can be see foaming, *muco-purulent,* bloody fluid becomes visible, a piration is forced up through the narrow passage with a If we are certain that no injury has been done with the the point of lesion, the expulsion of hemorrhagico-purule the stenosis confirms the diagnosis of carcinoma.

The pulsating motion of the esophagus, which is syn dilatation of the aorta, disturbs the investigation, partic of the disease be situated at the point of bifurcation. Bu tice, we become accustomed to this rhythmic motion of respiratory displacement of the organ, by which its wall another in expiration, scarcely interferes, provided bre deep and rapid; it is *absent,* or almost so, when *the can the walls* of the organ; it may, therefore, be absent up the entire circumference. On the other hand, there may disturbance due to severe cough, which, in spite of cocai a common consequence of the existing laryngitis or trach tant that the patients be examined upon an empty stom of vision is obscured by remains of food, such as portions of egg and the like upon the mucous membrane, and pa narrowed areas of the rough cancerous surface, mistakes in other cases it is easily possible to mistake these whitish

A single inspection will not always suffice for us carcinoma. In cases in which the diagnosis is uncertain, small particles of tissue may be extracted and later microscopically examined, but great care is necessary in this procedure.

The esophagoscope should never be used immediately after sounding with a rigid tube. If the sound has been introduced into the esophagus to determine a stricture and to measure its distance from the teeth, even with careful manipulation the field of vision may be obscured with blood. It is well, therefore, to wait a few hours.

The results furnished by *auscultation of the esophagus* are in the main meager. If the patient takes fluid, swallow by swallow, he himself will frequently notice, as do those about him, *a gurgling sound* which is sometimes heard for a few seconds and is produced at the narrowed area by the checking and regurgitation of the fluid stream. By auscultation at the previously designated area, these *deglutition murmurs* give us diagnostic points of support, which, although not absolute proof, are nevertheless auxiliary, particularly in those cases in which, for example, upon suspicion of aneurysm, it is unwise to introduce instruments into the esophagus. No importance is to be attached to the rarity of the murmur in question (pressing-through murmur) or its absence; if it be present, however, and conspicuously prolonged, an impediment in the upper portion of the esophagus is indicated. *If, however, the pressing-through murmur is invariably absent,* simultaneously with the first sounds—an occasional absence is without importance—this is in favor of a pathologic process which limits the musculature in its function at the cardia, and which, according to experience, is, as a rule, of a carcinomatous nature; for only very exceptionally are both murmurs absent under normal conditions. Rarely does the esophageal bougie, even when it does not pass into the stomach, fill itself with fluid or pappy or compact remains of food which must have lodged in that portion above the carcinoma; they betray their origin by the absence of hydrochloric acid, pepsin, bile, and peptone, and by their alkaline or neutral reaction, which, however, may occasionally be acid, and is then due to organic fermentative acids.

The *gastric function* must suffer sooner or later, and earliest if the cancer be situated in the vicinity of the cardia. The hydrochloric acid secretion is therefore not rarely found to be absent. The *urine* is scant owing to the limited ingestion of fluid, and, in advanced stages, frequently shows the products of proteid decomposition (indican) and those of pathologic albumin decomposition (acetone, aceto-acetic acid, oxybutyric acid). Examination of *the larynx, lungs* and *pleura* should never be neglected. This often gives valuable aid in judging our cases.

DIAGNOSIS

The most important point in the **diagnosis of carc** **esophagus** is the *recognition of a stenosis* of the esophagus. *mine this by the aid of the bougie in a previously healthy p* *the ages of forty and seventy, the affection having arisen w* *nizable cause, and steadily increased from weeks to months, n* *likely than that we are dealing with a carcinoma of the esop* stenosis that has developed in this way, and under these c occasionally be of a non-malignant nature, and on account tance of this point for the patient, this possibility should fir It must be borne in mind that the history and the subject as well as most of the objective symptoms, may lead us ast times happens, for I have myself observed and described s in benign dilatations and with the formation of a diverti as in spasm of the esophagus and atony, dysphagia may assignable cause, and increase in a few months to a thre rapidly undermining the strength. On the other hand, in the chronic nature of disease of the esophagus, we must by mit ourselves to give a relatively favorable prognosis, and cinoma. I have seen patients in whom difficulty in deglutiti from four to ten years prior to death, and due to carcino of the obstruction. These cases may be explained by the l the cancer formed secondarily upon a base which had long of irritation, and had been well prepared. We know that a variety of causes (to tuberculosis, to caustics, or to diges in cicatrices, in inflammatory, irritative areas, therefore, chanical obstruction causes local irritation, prepare a soil s formation of cancer.

A second factor in the history, which can only be appl ough investigation, is the age of the patient. It is true develops at an advanced age; but I have seen a number of of the esophagus in persons between 30 and 40 years of a a year passes in which I do not see at least one case in early

Another point which may mislead us is the *cachexia.* fest in *all* diseases of the esophagus, provided they form the ingestion of food; in nervous disturbances and in dil esophagus, it may even be excessive.

It must be remembered that a *steady increase of dysphag* or even for years, occurs in various affections of the esop in simple inflammatory processes, in dilatation, and in di conspicuous *change* in the power of swallowing is present *turbances* of the esophagus; but nervous influences may al appear in carcinoma.

Resistance to the passage of the esophageal sound is objective proof of importance in diagnosis, but this does not demonstrate with absolute certainty that there is an anatomical obstruction. *Spasm may arrest the bougie,* and it may be impossible to overcome this either by prolonged waiting or by attempts at swallowing on the part of the patient (see below). More important is the recognition of an *admixture of blood, of pus, or of the products of decomposition* which have lodged in the opening of the tube during probing, but it must be borne in mind that ulcerative processes and decomposition may be present in dilated portions of the organ without the presence of a carcinoma. Only when we are fortunate enough to find *shreds of tissue,* which unquestionably demonstrate the carcinomatous character of the affection, is this positive result a decisive proof; and this can only occur in exceptional cases; in the early stages of the disease when the diagnosis is still doubtful it rarely happens.

A *positive and early diagnosis* may be made most frequently by the *esophagoscope.* The esophagoscopic picture is generally the determining factor; in the majority of cases it may be readily obtained; in a minority this is unsatisfactory, i. e. it does not give a positive result; and this may be readily understood when we consider the fact that we are able to inspect only the upper border of the diseased area, and the later course of the affection and the consideration of all its accompanying circumstances can alone clear the situation.

From this it is evident that the positive determination of the character of a constriction of the esophagus is by no means easy. Although the malignant form predominates, many others must be considered which furnish a similar symptom-picture, and well deserve the interest of the physician. We should, therefore, avoid making a positive diagnosis of cancer of the esophagus, as is very commonly done, without certain indications such as dysphagia and resistance upon probing. Based on these criteria, no positive diagnosis is possible, and, when we consider how much is at stake for the patient, we should be somewhat more thorough in our differential diagnosis and more cautious in our prognosis.

We shall now describe the other manifold processes which cause obstruction to the passage of food, and, therefore, present the picture of stenosis of the esophagus. These are to be minutely discussed, and are best considered according to the classification given above. As benign neoplasms (myoma, fibroma, and cysts) are great rarities, and scarcely ever give rise to clinical symptoms, we may at once direct our attention to the *second group* of stenoses, those which originate from **inflammatory ulcerative processes.**

Stenoses of the lumen of the esophagus due to the *action of caustics* stand first in practical importance. These, as is well known, are not rare, and in regard to the symptoms it is immaterial whether acids, alkalies,

or other chemical agents, which have been swallowed eit
of poisoning or by accident, produce alterations in the mu
We shall not describe the stage of the acute, florid, inflamma
the phenomena to be attributed to this. We are more i
conditions which result in the course of weeks and month
of infiltrations of the wall, formation of cicatrices, and n
lumen. The strictures which originate in this manner,
annular or tubular, are limited to certain areas of the eso
is generally the upper portion, the region of the bifurcati
of the foramen œsophageum. If we are dealing with lon
ures, their centers are at these points, whence they distr
upward or downward or in both directions. There may
several points of the esophagus, and by the coalescence of
striction becomes extensive, and finally *total.*

The difficulties in deglutition are not specially chara
are permanent, and vary in intensity according to the de
striction and the local irritation, as is true of all steno
diagnosis is the circumstance that *from the history alone t*
disease may be determined with some degree of certainty. I
condition of the organ caused by caustic we can only ascer
investigation, and here our first question is: Are we deal
more strictures? In this respect, the reports of the patie
act of deglutition permit some deductions. However, t
must be utilized with necessary reserve, as it is frequent
erroneous. Moreover, we desire to know: Are inflamm
processes still present in the mucous membrane, or has d
tion occurred in all parts? Is dilatation present? Final
must be answered whether carcinoma has developed in con
changes which are to be here considered. Information i
these points is given us by *the sound* and *the esophagosco*
firm English and French bougies, also the metallic spirals
ably employ when it is necessary to find a passage, may be
in order to acquaint ourselves with the situation and to en
a correct opinion. The esophagoscope gives us accurate kn
ures due to caustics form very characteristic pictures. In
the suprabifurcating area of the esophagus frequently stri
and flaky white cicatrices are outlined sharply against the
membrane; the nearer the stricture the more numerous the
the mucous membrane. The ring form, or the beginning
stricture, manifests itself either as a cicatricial funnel at th
a more or less circular or oval lumen is noted, or it reser
vaginalis, from the fact that the region of the esophagus ab
and with the tube is uniformly forced downward above t
stricture. Only with a very superficial cicatricial formatio

rowed area, whose lumen is often eccentrically layered from contraction of the cicatrix and frequently resembles a folded diaphragm, still show respiratory movement and radial folds; the deeper the effect of the caustic, the more rigid and immotile is the area of the stricture. The whole portion looks like a canal partly or entirely covered with white cicatricial tissue. At other times the mucous membrane up to the point of stricture is a deep, dark red, and shows a tendency to bleed readily; in the earlier stages of the disease, and even later, or as the result of improper treatment, we find ulcerative areas between the cicatrices.

If the caustic action has been slight, the secondary cicatricial formation may be of little extent, and after the acute symptoms have passed away no difficulty in deglutition remains; but in several cases of this kind I have observed that 10 to 20 years later difficulties in deglutition may reappear, particularly if the food is very coarse; larger, poorly masticated particles may be arrested. Thus, in adults, I have twice been obliged to extract coarse pieces of meat, about the size of a thumb-joint, which were arrested in the upper third of the esophagus. In both cases slight corrosive strictures were present which had been formed in earliest childhood. Up to that time the patients had ingested their food without any difficulty, and scarcely remembered the early disease of the esophagus. In both instances, the esophagoscope revealed a marked injection in that portion of the esophagus in which the bolus was arrested. Only in one of the patients was there a distinct cicatrix. With care further consequences were averted.

Stenoses due to *other ulcerative processes* are very rarely multiple. Mostly limited to a portion of the wall, the contracting cicatrix draws the corresponding portion of the retained mucous membrane toward it, and thus the stenoses cause *diverticuli and valve formation.* It may then happen that if the bougie is caught in the narrowing, it may appear of great extent, while, at other times, quite a large tube may be passed with ease. The differentiation of cicatricial strictures according to the nature of the ulcerative processes to which they are due is very difficult, and even impossible except in the case of the ulcer due to caustics. We consider chiefly *syphilitic, tuberculous, peptic,* and *diphtheritic* ulcerations. In doubtful cases the presence of syphilis and tuberculosis will aid in the diagnosis; peptic ulcers occur only in the lower third of the esophagus, and there are usually accompanying symptoms which point to gastric ulcer. I treated successfully a case of stricture of the esophagus following scarlatinal diphtheria. Naturally here also the preceding disease is a guide to the diagnosis. In conclusion we must consider cicatricial constrictions which have their origin in *peri-esophageal* processes which start from the lymph-glands, secondarily producing ulceration of the esophagus, which, in case healing occurs, results in stenosis of the lumen.

The finer differentiation of cicatricial strictures according to their genesis is not of practical importance. It is, however, always an advantage to obtain a clear picture of the anatomic condition by aid scope, and thus determine whether or not florid inflamma present, and whether carcinoma, especially, can be exclude

Of great import is the recognition of stenosis due **esophagus.** By the pathologic contraction of a layer of t of the musculature, a segment of the esophagus becomes can only be passed with difficulty. The obstruction to deg from this is of varying duration and intensity, and occ accompanied by painful sensations. The spasm may be t manent; sometimes the affection persists uninterrupted months or even for years, occasionally it is intermittent, a only occurs after a definite irritation from food. In esophagismus *accompanies hysteria and neurasthenia*, epil tetanus; it is an invariable and most important symptom Occasionally it is one of the manifestations of disease of th system; very frequently it is *reflex.* Thus, a spasm may pı of our instrument, even of a soft tube; foreign bodies, co tive fluids, anything that produces decided retching, may born spasm. *Above all, spasm is frequently a concomit other diseases of the esophagus.* Every anatomical process may be accompanied by spasm. Diseases of *other organs* spasm; affections of the pharynx and larynx, of the stoma of the male and female genital organs, may be its eti *trauma,* which directly affects the head of the thorax, spasm. The appearance of spasm in certain *intoxicati* donna, from strychnin, from sausage, etc.), following r or without rheumatic affection of the joints and muscles, a out catarrhal affections of the larynx and pharynx, is worth

This neurosis shows itself chiefly by dysphagia. Th difficulty in deglutition is often not characteristic; the s same as those complained of by patients with stenosis from other causes. Occasionally the spasm is accompanied in the chest, by a painful feeling of constriction, by lancin pains in the neighborhood of the esophagus, for example, or the neck, or it follows these symptoms. Spasm of th pharynx, of the larynx, of the trunk, and of the extremity alternate with esophagospasm, particularly in those cases eral neurosis is the foundation of the affection. The deg in deglutition varies greatly. Only rarely is the esoph or almost wholly, impermeable for a long time; in these ca tion may result, and the patient finally perish from the dysphagia may appear in dissimilar forms. Sometimes perience only the sensation of an arrest of the food, and few deep inspirations, or by swallowing some fluid, the bolus finally reaches the stomach; or the first food is regurgitated while that subsequently taken passes without difficulty. In some cases the spasm shows itself only upon abnormal irritation, for example, during probing or when certain foods are taken, usually coarse food or solid food in general; but we sometimes meet a patient who finds fluids more difficult to swallow than solid food. Occasionally the spasm occurs only at certain times of the day, or during the course of prolonged meals.

In spite of the fact that some of these disturbances we have mentioned certainly arise only under the influence of a derangement of innervation, positive proof that we are dealing only with a nervous affection can never be attained in the given case by the history and the subjective observation of the patient. *The diagnosis of spasm is rarely easy.* The most useful points of support for the recognition of the neurosis from the subjective and objective symptoms will now be briefly described.

The sudden appearance of difficulty in deglutition, particularly of fluid food, indicates the presence of spasm, and still more so the *intermittence of the attacks,* which may be quite irregular or be produced by very definite influences, partly nervous, the intensity and duration of which vary. *Differences in the degree of permeability* of the esophagus is a conspicuous symptom favoring spasm; the first food may be arrested, the succeeding may pass, or, the first masses of food may pass readily, and then, without assignable cause, a sudden stoppage occurs. More important is a *variation in the seat of the constriction,* so that, for example, on one day the upper, and upon another day the lower, third of the esophagus becomes impermeable. But this is a very rare symptom, and is only found as an accompaniment of severe nervous affections. An absolute diagnosis of this condition can only be made by means of *the bougie* and *the esophagoscope.* The first sounding should take place with a rigid instrument, as this will more certainly pass the axis of the organ than a soft one, which is easily bent. If we probe during a time when the difficulty in deglutition exists, a *resistance* must be objectively noted. After a short pause, or on moderate pressure, it may disappear, for there are many mild forms of spasm, but it *must positively be present.* It is worthy of note that the resistance may be caused to disappear by probing, by means of which we force the patient *repeatedly to swallow very rapidly,* and this brings about a more decided innervation of the longitudinal muscles of the esophagus, therefore, the dilators, and thus we overcome at least moderate grades of spasm. Moreover, probing may reveal a variation in the degree of permeability and in the seat of the constriction which is often of decisive import.

By means of the esophagoscope we are enabled to recognize *other esophageal diseases,* and *positively to diagnosticate spasm;* for this, as a rule, usually gives a characteristic picture,—sharply rising folds of mucous membrane which converge toward a point in the middle of the lumen and form a rosette-like, more or less rigid closure. The lumen, if i at all, is narrow. Only upon deep inspiration or on c (mucus, gastric contents) and air regurgitated through ing, not rarely accompanied by a hissing or gurgling mur membrane of the contracted portion of the esophagus sl tense reddening. Occasionally the mucosa is found e in the region where regurgitation or stagnation has occu

If an examination be made during a time free fro previously mentioned criteria may be absent, but a n particular, is of diagnostic value. *The determination mentioned causes of spasm* is an important aid: *The affection of the esophagus coexisting, the recognition c of which the spasm of the esophagus may be the reflex* all, we must consider the status of the nervous system ease of this region be unquestionably present, it does clusion regarding the character of a coincident difficu This must be borne in mind if we would avoid fallin Inversely, we should observe that in true spasm als deglutition may progressively increase, that extreme develop, and that more or less complete impermeabilit may persist stubbornly for months and years.

Congenital stenoses of the esophagus, from the onse impossible, or they manifest themselves by a difficulty i appears in early youth; it must be remarked here that c of the organ (anterior stomach) may give rise to sympto of stenosis. If it is possible to demonstrate that cons food are retained in the esophagus, this is proof that is present; whether beneath this a constriction also determined by the sound and the esophagoscope.

Occlusion stenoses, to the description of which we to impaction by *foreign bodies* which are arrested in tl occlude the passage more or less completely, irritate the and eventually may produce phlegmons and perforation. objects, for example, small fish bones, by giving rise to spasm, may cause pain and symptoms of stenosis; and, body is not arrested but reaches the stomach, *erosions* it produces in the mucous membrane may cause the sa the contrary, it will be observed that not only small but *large foreign bodies,* for example, a plate of false teet in the esophagus *without giving rise to decided subjecti objective, symptoms.* For example, as I myself have se teeth larger than a silver half-dollar may be arrested of the esophagus below the larynx, and lie with its convex surface in the concavity of the anterior wall of the esophagus, so that the organ gapes, and thus not only food but thick bougies may pass the large foreign body without difficulty. In this case, it had been supposed that the plate of false teeth was no longer in the esophagus, and the symptoms still present had been referred to lacerations in the mucous membrane; the true state of affairs was revealed by the esophagoscope.

Foreign bodies, almost without exception, are arrested in the *upper,* narrower parts of the esophagus; when they are found in the lower portions they have usually been forced down by instruments. With a previously existing stenosis even a small foreign body, for instance, a fruit kernel, may bring about complete obstruction of the esophagus, and absolute impermeability arises when a spastic process of the musculature holds a small foreign body.

If foreign bodies have been swallowed, the patient is generally aware of it, and reports the circumstance; where no *history* can be utilized, *the acute appearance* of stenosis at once leads us to think of obstruction; but also when the stenosis is due to foreign bodies, the *variation in permeability* occasionally favors this hypothesis. Temporarily the closure is complete, then the foreign body, in consequence of a paroxysm of coughing, or by retching, changes its position, and food now passes, perhaps also a bougie. But the sensation of a foreign body being lodged in the esophagus mostly remains, and after a short time difficulty in deglutition again becomes noticeable.

The examination with the bougie in doubtful cases will scarcely show the nature of the obstruction. By the introduction of instruments, we are at most only able to determine that a mechanical obstruction is present, and this is not even certain in all cases; for the probe may pass alongside of the foreign body through the lumen of the esophagus. On the other hand, *esophagoscopy always enables us to make a positive diagnosis.* By the aid of the eye we can extract the foreign body, or force it down, and readily cure any existing lesions in the mucous membrane by cauterization.

The **stenoses due to compression of the esophagus** are of the greatest practical importance. Any tumor formation in the neighborhood of the organ may sooner or later lead to compression of the tube. The esophagus is naturally motile, and may deviate. If, however, it is completely surrounded, or only adherent on one side, symptoms of stenosis appear. *Goiter* itself most readily causes difficulty in respiration, but rarely disturbs the function of deglutition; this will occur if it be very large, or if it surround the esophagus with greatly developed processes, or show malignant degeneration. Carcinoma of *the larynx* or of *the vertebral column* often interferes with the act of deglutition, and very early.

In the thoracic cavity, as well as in the neck, the esophagus may deviate

produce a favorable influence upon nutrition. Nourishme
be *plentiful,* and, under some circumstances, it should be
crease flesh. It need scarcely be mentioned that in the
problem great, frequently insurmountable, difficulties are c
here much may be attained by the proper choice of food, a
tion of all auxiliary methods for artificial nutrition. W
a rule that the patients take *only such food as we know*
through the narrow space; for it is possible to take suf
though it be in the thinnest of fluids. Trials with coars
cases, only increase the local irritation, and thereby make
to swallow liquid food. *Large particles may be arrested,*
the stenosis completely impermeable; connective tissue sh
lar, which adhere to particles of fat and meat, obstruct wi
ease a lumen which is already narrowed. For this reason
the greatest care to have the particles of meat, vegetable,
possible, and where we are not certain that the patient ma
thoroughly with his teeth, these, so far as permitted, shou
in a crushed form. That foods with skin, stems, kernel
forbidden is self-evident, but even *highly seasoned* or *spice*
etc.), concentrated alcohol, or very acid foods, are to be av
of their irritative effect. The same is true of *extremes*
Lukewarm or slightly cooled fluids are easiest to swallo
not contradict the fact that occasionally, where there is m
and swelling, ice may be well borne; food of a low ter
suitable for prolonged use, as it unfavorably influences ga

Naturally, the *food in its composition* should contain *c*
nutritive elements, and as we are able to administer not onl
hydrates but even fats and proteids (the latter, for exam
of artificial foods) in a form soluble in water, it is quite p
the patient sufficiently in those cases in which only a thin
attach especial importance to the administration of *fat* in th
butter, or olive oil, or cream. Apart from the high com
which it is distinguished above all other food, it is parti
because it makes the bolus soft and slippery, and beca
the mucous membrane, it protects the same, thus dimini
bility of the inflamed parts; sometimes it will trickle thr
when even water is regurgitated.

The arrangement of meals and the manner in which t
of vital importance. All over-exertion of the diseased
avoided. The patients are therefore advised to swallow *slo*
between; where compact food is taken, the ingestion of thi
bite will facilitate its passage. Each meal, therefore, sh
comparatively long time, even when only small amounts o
but this does not matter; such patients should be encour

often. That mental rest and bodily relaxation are beneficial here is true, all the more so as unfavorable nervous influences very readily aggravate the disturbance in swallowing, even in those cases in which a carcinoma is present. The avoidance of all unnecessary exertion is absolutely essential for the purpose we have in view, namely, to improve the nutrition. Not rarely we observe that with complete rest in bed the power of swallowing improves. It need hardly be mentioned that everything that stimulates the appetite and increases the power of assimilation (fresh air, massage, wine, etc.) will aid in the treatment.

Further curative measures in patients with stenosis of the esophagus depend upon the nature of the underlying process. Let us first consider the treatment in the cases which are in the majority, in *carcinoma.* The object in cancer is this, to maintain the patient in such a condition that he is able to ingest fluid or pappy food readily, or, at least, without decided difficulty. We attempt to keep the stenosis from becoming impermeable, but it is not our aim to extend our therapeutic endeavors beyond the limit just mentioned, and to bring about greater ease in swallowing more compact food no matter at what cost. If it is clear to us to what the dysphagia in cancer of the esophagus is due, the two possibilities which are present here, and to which I have already called attention, will, above all else, prevent this. In the first place the disturbances in deglutition are produced by a *mechanical obstruction,* by infiltration of the walls, or by the proliferating neoplasm. The second factor of no less consequence is the damage to the *nervous* and *muscular apparatus* of the esophagus, which, in some of the cases, is in an extremely irritable condition, and reacts abnormally as soon as food reaches the ulcerated, inflamed area. This reaction frequently manifests itself as *spasm,* during which not a drop of fluid will pass the stricture, yet the same patient, after a preceding hypodermic injection of morphin, may readily swallow solid food. At other times *atonic* conditions react unfavorably upon deglutition in that portion above the cancer, and even in the areas in which there is no dilatation. From hyperesthesia of the inflamed parts, by over-exertion, etc., irregularity and weakness of function may arise and lead to an interruption, or, at least, to a disturbance of peristalsis which manifests itself by an unfavorable influence on the muscles, sometimes the longitudinal, at other times the annular, the final result, however, being the same, namely, a disturbance of deglutition.

If we observe these points, we must distinguish the treatment of mechanical obstruction from that of irritative conditions and atony. The former is a very unsuitable point toward which to direct our treatment; in general more harm is done here than good, yet all the more must we attempt to relieve the *local irritation and weakness.* Besides a bland diet, we must prohibit any food that does not pass easily, anything that may

cause regurgitation, and must consider an anesthetic p
administered internally is often beneficial; the objection
use (becoming accustomed to the poison, and loss of appet
not slight. Morphin may be administered alone, or in
cocain or antipyrin or menthol in the form of compres
are slowly dissolved in the mouth; I use these very fr
seated carcinomata which are near the larynx and pharyn
these organs (for example, morphin muriate 0.0025, co
pyrin 0.1, sacch. 0.3. M. f. tabl. compr. dos. 30; one ta
daily, immediately before eating).

Where morphin fails of success, I resort to *local anes*
aid of the esophageal syringe devised by me (a simpl
capacity of about 3 grams, and to which a fine tube of al
in length is attached) I inject, reaching the point of the
to five per cent. *eucain solution,* at first twice daily, late
doses of from 2 to 3 grams of the solution per day are
not give rise to symptoms of intoxication. There are a
cauterization of the hyperesthetic inflamed mucous mem
per cent. silver nitrate solution may prove beneficial.
is sometimes remarkable, even though, for the most part
but, nevertheless, this process assists materially in att
sketched above, namely, the retention of the patient's
fluid and pappy food. The process of injection is quite
be employed by any physician; it can never do harm, bu
same may be said of mild measures to combat atonic and
—here, above all, mild *lavage of the esophagus* with the
which we employ for the stomach is advisable and re
mucous masses, obstructing coagula, remains of food, forei
of decomposition, etc., are thus removed, and, simultane
ing process acts also as a kind of massage, and has a
the muscle.

All of these auxiliary measures are more worthy of
ment of the carcinoma than *probing,* by which the mech
which causes the disturbance is increased. In my experi
of all malignant neoplasms are unsuitable for treatme
and only in a few cases have I been convinced of an impro
tion by this process. *So long as the patients are able to*
pappy food, I avoid every process which dilates mecha
cases we must be content with the existing condition, f
of attaining anything more satisfactory by sounding is ve
ability to swallow is markedly disturbed, so that degluti
is only accomplished with extreme difficulty, if anesthet
the condition, if lavage simultaneously is without result,
refrain from dilating the stricture. It must be borne in

ing is never an insignificant procedure; it increases the irritability of the organ, the tendency to hemorrhage and ulceration, and adds to the danger of perforation.

In probing, the hollow, fenestrated, hard rubber instruments may be used with advantage after being previously softened in warm water, but, nevertheless, by bending in the stricture, or even before they reach it, these are soon worn out. Solid bougies of the same material with a button-shaped or pointed end are more satisfactory, although they are not much more durable. I have for years frequently employed bougies which were manufactured for me after Crawcour's method. They are made from rolled sheet metal, cut into spirals, the flexible part, 40 cm. long, making up the coils; they terminate in a button-like attachment about 1½ cm. in length, the upper part consisting of a solid steel handle 10 cm. in length. They are very flexible, find their way into the stricture even if it is excentrically situated more readily than other sounds, and are almost indestructible if kept clean (a diluted lysol solution and subsequent drying by heat are sufficient for cleansing purposes). In carcinoma we must be extremely cautious in the passage of the sound, and never employ the slightest degree of force. The sound, which will barely pass, is allowed to remain for a few minutes; it should be introduced every other day, not oftener, for the diseased mass reacts to every irritation, bleeds readily, and thus edema and pain are rapidly produced. Often it is difficult to reach the narrow canal from the dilated portion above the stenosis, especially as this passage is not always straight; portions of the tumor may occlude the opening, or the sound may bend and be pressed against the wall of the organ, where even slight pressure may be followed by the most serious consequences. If the opening into the canal is not readily found, further manipulation should be stopped for a time; if, however, we succeed in passing an instrument of considerable size, at the next trial we should try most carefully to pass one a size larger. If we succeed in this, a still larger one may later be used. In the main, we must be careful to use no force in this method of treatment, but should rather be content with moderate success, and avoid too frequent and too prolonged soundings.

It will not rarely surprise us that although, for example, No. 5 passes readily, at the next attempt No. 2 can hardly be passed through the obstruction; acute swellings have appeared, or the tumor, in consequence of irritation, shows a more pronounced growth into the lumen of the canal. On the other hand, it may happen that the narrow passage will suddenly become permeable for compact food and thicker bougies owing to the removal of obstructions by ulcerative decomposition on the part of the tumor masses. If it is impossible to introduce a bougie in the usual manner through the narrowed esophagus, the attempt may be made with the patient in the recumbent posture, eventually *with the aid of* the *esophagoscope;* instead of working in the dark we may occasionally find the entrance to the canal immediately, and we may sometimes succeed in cases whe viously failed. For this purpose I employ spiral bougies English bougies with a solid metallic guiding staff.

For dilatation of esophageal stenosis, still other instru employed. Senator was the first to introduce *laminaria* thickness into the carcinomatous stricture to attain a dil gradual swelling. The laminaria tent was fastened by a flexible bougie. I have entirely abandoned this method of carcinoma; but the idea may be well utilized in the dil strictures if the laminaria tents armed with a silk thread tion of the eye and by the aid of the esophagoscope are the narrow passage, and are permitted to remain from thr perhaps even longer, and the tents are then drawn up by Dilatation may thus be promoted with extraordinary rap soon convince ourselves that there is no danger in this pro perate cases I have obtained from it excellent results (see

To produce a slow and careful dilatation, Schreiber a consisting of an ordinary, thin, non-fenestrated stoma esophageal end of which a rubber tube 2 to 3 cm. in len which again terminates in a smooth firm point. At the the sound there is a metallic attachment which is connected syringe having a capacity of from 10 to 30 c.c. The sou through the narrow passage, water being injected into th the rubber piece fills and expands. The tense, elastic attempt to withdraw through the narrow passage. This, tremely difficult, but with great care quite decided pressure the internal walls of the stenosed area. This process I ver in carcinomatous strictures, as they are generally too unyiel to the pressure. But the method is well adapted to soft, r tissue, and to overcoming spastically stenosed areas.

[Professor B. W. Sippy, of Chicago, has invented an the dilatation of spasmodic and other strictures of the esop cardiospasm (see Fig. 1). The instrument is especiall hypertrophic stenosis of the cardia due to long standing c has been successfully employed in several patients. In tw my charge the result was notable. In one, a woman wit cardiospasm and apparent hypertrophy of the circular fil spasm of the cardia, and with considerable dilatation of the applications of the instrument resulted in complete relief. failed after a trial of several months. The instrument is ap of organic stricture, but should then be used with extreme

In conclusion I must mention the treatment with a *per* which was so strongly advised in foreign countries, was te and Renvers, and has been of late especially praised by Cu nnula employed for this purpose is best manufactured from elastic material. it should be 10 to 12 cm. in length, and have at its point a width

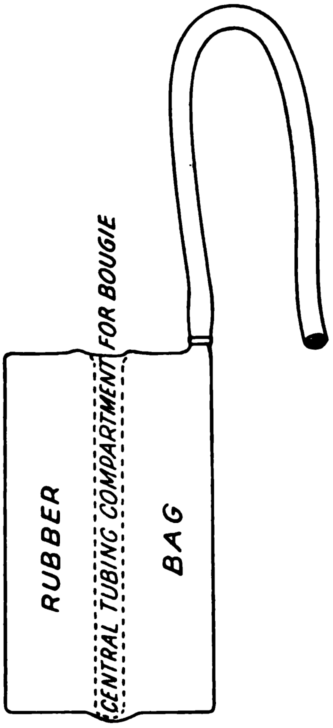


FIG. 1.—The Sippy Dilator.

[The dilator consists of a rubber bag 3 inches long and 1½ inches wide when col- d. At an upper corner of the bag a firm piece of rubber tubing about 20 inches is attached. Another piece of rubber tubing 3 inches long is secured in the center e inside of the rubber bag. The lower end of this piece of tubing is closed; the er end remains open and is secured air tight in the wall of the rubber bag. The tip of a bone bougie introduced into this compartment guides the bag into the seat of the ture. The essential feature is a bag made of thin firm cloth which encloses the rubber and limits accurately the degree of dilatation. The length of the cloth sac should be at 3 inches. Its width determines the degree of dilatation. In dilating strictures o spasm the width of the cloth sac should be about 6 or 7 cm. for adults. ller sizes must be constructed for children and for dilating strictures due to malignant tis and cicatrix. The cloth sac must be uniform in width. When ready to introduce, ther condom is slipped on over the collapsed dilator and tied loosely about the rubber ag and bougie. The distance of the stricture from the teeth is measured and the collapsed ring bag guided well into it.

A firm rubber bulb such as accompanies an ordinary Paquelin cautery may be used as air-pump. The dilating force that may be applied directly to the stricture is enormous, limited accurately by the size of the cloth sac.—ED.]

lumen of about 5 mm. and above of about 12 mm. Such a cannula ntroduced by means of a whalebone stylet with two ivory buttons; the er button serves as an obdurator. The upper is larger, and permits

the introduction of a tube which is held by means of a si
the stylet has been withdrawn the thread is fastened upon
advantage of this process is that nourishment, either flui
may be introduced in quite large amounts without diffi
said to prevent the decomposition of the cancer, as the fo
in contact with the tumor. My experience with the u
cannulæ is not particularly favorable. The retention of t
the mouth is uncomfortable to the patient; if it is drawn
it irritates the epiglottis and the pharynx, and is most un
patients, for example, if catarrh of the larynx or of th
is so frequent, is present. The mechanical irritation of
also not to be underestimated. Encircling of the tube b
clogging of the same, are not rare. The most unpleas
ever, is this, that occasionally in withdrawing the cannula t
and we are then compelled to remove it with the esopha
may be a very difficult operation. Above all it must be
this cannula treatment is only adapted to patients in who
5 to 6 mm. still passes the obstruction. There are, how
whom for some time sufficient nourishment may be intr
to the method I have first described, without subjecting th
discomfort. The methods with laminaria tents and with
sound presuppose a certain permeability of the stricture,
the extent necessary for the cannula, but this is evidently c
why these methods have not been more frequently resorte
ment of carcinoma.

If the stricture is too narrow, so that probing with
esophagoscope gives no results, and the local application
bat the hyperesthesia and the spasms affords no material re
fore, the condition is such that, in spite of all endeavors,
slight amounts, trickle through the esophagus, and the
emaciate, in my opinion the time has arrived for *surg*
But before deciding upon this, I would urgently advise
of putting the patient to bed for a few days, with abso
nourish him by the bowel, entirely refraining from the int
by the mouth. Hunger and thirst may be allayed by sma
(0.02 per dose, two or three times daily), and the mouth an
be frequently rinsed with ice water. A favorable chang
in that the local condition improves decidedly. After th
rest, the passage is likely to be decidedly more free than
and the introduction of the tube or the injections may b
this appear advantageous; sometimes it is unnecessary,
if such patients are nourished with enemata on one or two
they are able to swallow quite well during the remaining
larly if small doses of morphin are simultaneously admin

stage of the disease we have little hope of long averting the catastrophe, but whether in this crisis gastrostomy will greatly avail appears to be very questionable.

In my opinion, only in the most unfavorable cases is the production of a gastric fistula indicated. This operation is, in truth, for me the *ultima ratio*, notwithstanding the fact that the operation, through the successes of prominent surgeons, has become a relatively safe one, and that the fistula functionates perfectly for a long time. *Gastrostomy* appears to be absolutely indicated when *broncho-esophageal fistulæ* cause the continuous passage of food into the wrong channel, when imperfect permeability of the esophagus for any kind of food exists, and, finally, when in an early stage of the disease impassibility occurs and causes a rapid loss of strength. In well-advanced cases, with decided stenosis of the lumen, a marked prolongation of life, a noticeable improvement in the general nutritive condition, and a decided amelioration of the dysphagia is not to be expected from the operation. As a prophylactic measure there is not the slightest reason for early operation while there is still sufficient permeability of the esophagus. The *radical treatment* of cancer of the esophagus by extirpation could only come into question with high-seated tumors; up to this time not a single case of recovery has been reported. This is partly due to the great difficulties which the surgeon must combat, but partly also to the fact that the diagnosis is not made early enough. The more thorough use of the esophagoscope may, perhaps, be productive of better results in the near future.

Of other *symptomatic treatment*, I shall only mention the irrigation of the esophagus with disinfectant solutions (thymol 0.5–1000, salicylic acid 1–1000, silver nitrate 1–1000, etc.) to remove the symptoms due to stagnation (fetid odor, eructations). For the catarrh of the pharynx, of the larynx, and of the bronchi, which so frequently accompany the condition, I employ Ems water, morphin, etc.; the salts of iodin, also, bring about amelioration by thinning the tough mucus. The many neuralgic pains are relieved by our reliable antirheumatics.

The therapy of *cicatricial* strictures must be constructed on principles entirely different from those that come into question in carcinoma. Here there is a possibility of preserving life, under favorable circumstances of permanently curing the local lesion, and here *dilatation of the stricture* is the most important aim of the treatment. The results depend very much upon the methods of procedure. We shall first describe the simplest process, that of *sounding*. It is generally admitted that *slight* pressure is permissible in shoving the instrument through the narrowed cicatrized area, and that it may be permitted to remain for some time (from three to fifteen minutes). Daily probing is not always advisable; often a certain amount of forbearance gives better results, so that the method is only employed

every second day. If we are dealing with an excentric
or with a somewhat circular narrow pass, the metallic
as I advised many years ago, appear to me to be the
struments. If the way into the stricture may be foun
is quite immaterial of what the dilator is made. Sound
whalebone, or English or French bougies, may be employe
sible to attain our end by the usual sightless method, b
upon further surgical procedures (esophagotomy, gastro
always make an attempt to probe with the aid of the e
apparently quite desperate cases I have at last succeed
entrance to the stricture, and in passing it with a fine
process is laborious, but the results are sometimes brillian
with the esophagoscope, I am partial to the use of meta
or so-called "director sounds," i. e., narrow bougies of i
which terminate in a quite long metal staff that permits
Occasionally I have also employed with advantage fine le
to direct the passage through the obliterated organ. Aft
a few times with the esophagoscope, I frequently succee
purpose upon simple probing in the recumbent posture
duction of the esophageal tube, and if the canal has b
I resume the trial in the sitting posture with the or
number of little artifices, which I do not care to describe
tate this process in difficult cases.

If the cicatricial tissue is not too firm, and the narr
long, I find Schreiber's sound, cautiously used (see ab
useful in producing dilatation with comparative rapidity.

The treatment of cicatricial strictures by bougies sh
early as possible. This treatment should be carried ou
should be continued for a long time, for, even after ext
the tendency of cicatricial tissue to contract continues.
attempt decidedly more in this affection of the organ th
cautious manipulation is nevertheless necessary, and it
mind that, besides cicatrices, ulceration may also be
mucous membrane above the narrowing is frequently
flamed, so that here, as well as in cancer, the distur
tion does not necessarily depend upon the mechanical
but may be due also to nervous disturbance, to a te
and to atony.

There are, however, cases where the most persistent
even if it really pass through the constricted area, produ
dilatation; and we often meet with cases in which it is i
the stricture with any of our instruments. In difficult c
and sometimes in almost desperate situations, an attempt
dilate in the esophagoscope with sounds or by the introdu

tents at the starting point of the stricture. The latter process, which Ebstein has advised as a modification of the earlier Senator method (which see), I have repeatedly tested. For the introduction of the stylets into the stricture I employ forceps with smooth, concave, internal surfaces; if the entrance to the stricture is extremely small, the lower end of the tent is pointed, and cautiously pressed into the narrow canal. During this treatment the patient must be very carefully watched. At first the tent should be allowed to remain only for three to eight hours, provided there is no pain or fever, and later, for a longer time. Withdrawal is usually easy by the aid of the silk thread. In elongated strictures the process with the laminaria tent is an arduous one, and gives only very slow results. In these cases we should always try to attain our end by the introduction of long thin bougies by the aid of the esophagoscope. After we have found a passage, according to v. Hacker and Ebstein, dilatation may be facilitated under some circumstances by employing drains which are placed over a thin metallic introduction rod of about 2 mm. in size; if the staff is withdrawn the tension lessens, the drain contracts, and thus exerts decided pressure upon the wall, particularly if it is allowed to remain for a longer time, up to 24 hours. In my own experience the process is somewhat painful and not absolutely harmless; even with close watching of the patient, the jerking back of the rubber may occasionally cause a laceration in the wall of the organ.

If we do not succeed by these methods, if the results are unsatisfactory in that deglutition is not facilitated, and the patient's nutrition now suffers, *gastrostomy* should not be too long delayed. By producing a gastric fistula, we need no longer be anxious about the nutrition, we may influence favorably the irritation and inflammatory condition created in the esophagus by the inactivity of the organ, and may devote ourselves to the object of restoring its permeability. For this time is often necessary, much time. Besides, there is the added advantage that through the wound produced by gastrostomy we may attack the focus of the disease in the esophagus. Primarily this is possible by means of bougies, and cases have been reported where dilatation has been produced from below at a point which could not be reached by sounding through the mouth. This presupposes that the fistula is located in such an area that from it we may with comparative ease reach the cardia. This, unfortunately, is not the case with the fistulas commonly produced by Witzel's method; their advantage consists in their permitting an excellent occlusion, but manipulation with sounds from the gastric opening is exceedingly difficult. There are methods, however, which, even under these circumstances, may be quite well employed for dilatation. The simple experiment, after Socin, with *silver balls* which are fastened to a silk thread should first be tried. A very small one may be swallowed by the patient in the evening, and we are sometimes surprised to find that in the course of the night it has found its way into the stomach. Various processes have been devised for securing through the fistula the silver thread which has been fastened above through the cheek. The stomach may b filled with water or soup, and these fluids are then permitted rapidly t flow off through the tube: not rarely the thread will be carried with them Or slightly curved instruments (dressing forceps) must be introduced int the stomach and an attempt be made to catch the thread while the patien assumes different positions, and to draw it into the fistulous canal. If thi is successful the ends of the silk thread are fastened together, the ring i closed, a fine rubber drain is inserted within it, and an effort is made t draw this through the stricture. Then larger drains may be employed, an the stricture be thus dilated. But we must not be too hasty, and too forcible pulling upon the thread must be avoided, as this causes great tensio in the narrow pass, and the danger of injury is not inconsiderable.

It must be mentioned that *esophagotomy* may also be occasionally en ployed if it can be carried out *below the obstruction.* Nutrition is possib through the fistula, and, under some circumstances, dilatation may l attempted at the opening in the neck. Here, however, it must be observ that a stricture in the opening at the neck of the esophagus may be dia nosticated positively, but it is by no means certain that there are not al other impermeable ones in the lower part of the organ. Then we ha to deal with a new obstruction. This we may occasionally remove fro the wound in the neck by probing, or cutting, but such a process is alwa protracted, and, in the meantime, the exhausted patient may peris Therefore in all doubtful and difficult cases it is wise to assure ourselv of the nutrition by the production of a gastric fistula. In the treatme of *compression stenoses,* sounding is also useful, and generally still mo so than in cicatricial constriction, for we may manipulate the organ to greater extent, the mucous membrane being everywhere intact.

We are soon convinced that a decided improvement in deglutition not to be brought about by bougies; but if, at regular intervals, we intr duce sounds with fenestrated openings into the stomach the nutrition the patient may be carried on satisfactorily, and thus inanition be prevent This is all that we can really accomplish in patients with compressi stenosis, if the treatment does not diminish the size of the tumor whi causes compression or remove it. The latter, unfortunately, is only exce tionally possible; for example, in syphilitic lymphomata by potassium iod or an inunction treatment, in diverticulum by cleansing or extirpation the sac.

In spastic contracture of the esophagus, soundings may occasional be of use; I have frequently brought about a lessened tendency to spasm the cardia by over-distention of the esophageal orifice of the stomach, f which Schreiber's rubber balloon sound serves the purpose best. This i strument is carefully introduced into the cardia, perhaps by the aid several attempts at deglutition, or by local anesthesia, when the balloon distended, and pulled through the cardia. Above all, however, we must endeavor to ascertain the cause of the spasm, whether inside or outside of the esophagus, whether produced by local disease, or whether the general condition produces the tendency to spasm. The etiological therapy which results from this is of vital importance, but, for this reason, the importance of the local symptomatic treatment is by no means to be undervalued. Besides sounding, the injection of remedies that produce anesthesia (eucain, 3 per cent.) or electricity by the aid of a simple esophageal electrode, such as I have proposed, may be of decided benefit. Where the spasm, particularly at the cardia, has produced secondary changes (dilatation, inflammation), the resulting conditions are naturally to be also considered in treatment.

In conclusion, a few remarks may be in order concerning the *treatment of obstruction stenosis;* polypus (an extraordinarily rare occurrence) should be removed surgically; in obstruction due to masses of thrush, irrigations and the use of borax (3 per cent. solution, a tablespoonful every two hours) internally, are very serviceable. When *foreign bodies* are lodged in the esophagus, we must above all things refrain from at once employing the sound. We must first endeavor to remove the object which has entered the esophagus by the mouth, particularly if it has pointed or sharp edges, for, if forced downward, these may give rise to dangerous symptoms in the stomach and in the intestines. Smooth bodies, for example, coins, even if large, are not very dangerous in their journey through the digestive apparatus. It is, however, of the utmost importance that (if possible) a foreign body be speedily removed, provided we may extract it under the direction of the eye. Esophagoscopy enables us to achieve this in many cases by a bloodless process, where formerly an operation was necessary; small pointed objects, particularly, are thus rapidly removed and without danger. Instead of the manifold instruments which were formerly necessary (coin catchers, hooks, esophageal probangs) we now, in most cases when employing the esophagoscope, use only a simple forceps. Combined with this an instrument constructed according to the principles of the Leroy curette may sometimes be useful. That a small foreign body which causes disturbance of deglutition may sometimes be removed by the administration of an emetic must be also mentioned. *Fissures and erosions* which subsequently cause difficulty are best treated by the application of caustic under the direction of the eye, provided rest of the organ and morphin do not bring about rapid recovery.

If the foreign body reaches the stomach, the old efficacious potato treatment for its expulsion per anum is still the best method: For three or four days the patient is given daily 3 or 4 pounds of potatoes prepared in different ways, constipation being induced by the administration of the tincture of opium; then the bowel is cleansed by enemata or the administration of castor oil.

THE HISTORY AND CLINICAL INDI OF GASTRIC LAVAGE

By W. FLEINER, Heidelberg

At the turning point of the century the treatment of has reached a high degree of development. This progress upon the fruitful soil of an exact method of diagnosis bas indications, and the seed was the ingenious idea of Kussma about a lifetime ago: Introduce a tube into the stomach, normal contents by washing, and treat locally the diseased membrane.

Up to the middle of the previous century, but few o changes of the stomach could be recognized with certainty for which pathologic anatomy had already furnished an ex knowledge of functional disturbances of the stomach was ev Digestive disturbances whose organic foundation was unre attributed, according to the views of the time, to patholo the stomach, and were thus treated.

Naturally empiric treatment of an assumed affection co unsatisfactory results—such as we sometimes attain even greatly eulogized remedy is used to relieve a prominent clinical case.

The modern physician who has been trained to employ according to definite indications and with clear ideas poss out effort on his part, an advanced technic which is the f of his predecessors; he can scarcely realize the conditions in modern auxiliary measures did not exist, yet in these we ha to the fullest extent. And he will correctly measure and the value of his inheritance only when he studies its histor and considers the labor that has brought to completion so

THE HISTORY OF GASTRIC LAVAG

At the close of the sixth decade of the preceding centu the stomach, particularly that advanced form developing and closure of the pylorus, was reckoned among the disea to treat. " Only exceptionally," says Kussmaul, " did we a in the treatment of this distressing affection. As a rule, little could be done to ameliorate the sufferings, and nothing to cure."

Cases of this kind were looked upon as hopeless, and for this reason were not welcomed in hospitals and clinics. Thus Kussmaul, at that time Director of the Freiburg Medical Clinic, was disinclined to admit to his Clinic, which was then greatly overfilled, a country girl, Marie Weiner, from Heimbach, aged 25, who sought admittance upon April 15, 1867. She had been suffering from a gastric affection since her eleventh year, having an extreme dilatation of the stomach due to pyloric ulcer, hypertrophy of the pylorus, and chronic catarrh of the stomach; she was greatly emaciated and suffered besides from peculiar spasmodic attacks which are now known and dreaded under the name of gastric tetany; however, at the repeated urgent entreaty of the patient, Kussmaul relented and admitted her to the Clinic.

This patient suffered almost daily from gastric pain and vomiting, and passed sleepless nights of suffering unless relieved by the administration of morphin; her pitiful condition gave Kussmaul the idea of employing the stomach-pump. His considerations and reasons were as follows:

"Quite frequently, when I observed the patient in the miserable prodromal stage of vomiting, the thought had occurred to me that I might ameliorate her sufferings by the employment of the stomach-pump, as the removal of large masses of decomposed acid gastric contents should cause the agonizing burning and retching at once to cease. The introduction of the esophageal sound was naturally not difficult, for where a gastric dilatation has existed for so long a time the esophagus also is usually dilated. The artificial emptying of the stomach by the pump could be no more painful or distressing than her condition before and during vomiting. At all events, it would be more rapid and complete than the natural emptying of the stomach by the act of vomiting, with its prolonged prodromal stage of nausea, pain and retching. Repeatedly, even after vomiting, palpation and percussion revealed that the stomach still contained considerable masses. This condition reminded me of the so-called ischuria paradoxa, in which large amounts of urine flow daily from the dilated urinary bladder without its actually becoming empty and without reducing its circumference. By means of the pump we must succeed in emptying the stomach *completely*, and, if its elastic and contractile power have not been entirely exhausted, perhaps even *give to it the tone to contract to its smallest extent,* just as the catheter occasionally brings about recovery in ischuria paradoxa.

"In our patient the gastric dilatation was due to constriction of the pylorus. At the autopsy of cases of extreme gastric dilatation I had repeatedly observed that the stenosis which caused them would still permit the passage of a small finger from the stomach into the duodenum, although toward the end of life there had appeared to be complete closure of the pylorus. In such cases I had occasionally noted at the bedside through the abdominal covers active movement in the stomach. A gastric musculature might be present, but certainly not com It appeared to me *as though the excessive distention, the fi loading of the stomach itself, produced a mechanical action* 1 *the constriction of the pylorus to complete closure,* and tl hoped to remove by emptying the stomach and decreasing it

“ Finally, it appeared that the employment of the stoma permit a *more active topical treatment of the diseased gastri brane* than was formerly possible. In the case of our pat had for years been continuously irritated by extremely acid retention, stagnation, and decomposition of masses of food because of pyloric stenosis is certainly often the only cause of the mucous membrane; as, for example, in cases in w there was but an ulcer or a cicatrix at the pylorus, the stom wise intact; in all other cases it probably maintains and inc already present. This is probably why we note so constan tion of the pylorus, that the mucous membrane about the the gastric contents especially accumulate, shows most inten stomach-pump, I hoped, would not only make it possibl evacuate these acid, irritating masses, but would also perm and cleansing of the diseased mucous membrane which ha by acid and alkaline fluids, as, for instance, with Vichy w artificial soda solution.”

In pursuance of these considerations Kussmaul, upon for the first time, pumped and washed out the stomach of

“ The introduction of the stomach-tube, the pumping ou ing with Vichy water were unexpectedly easy. We withdre acid, dirty-grey, sarcina-containing fluid, with particles of undergoing softening and decomposition.

“ Even the immediate result of the first emptying and stomach with Vichy water was a surprisingly beneficial on who was previously always exceedingly disagreeable, and of disposition that she well bore out her name, ‘ Weiner ’ (cr few hours later as if completely transformed. For the fi agreeable and appeared comfortable in her bed, and she d years she had not been in such good spirits. She at on slept much better, and for two days was entirely free from sations in the stomach. After repeated employment of tl end of fourteen days the patient had a more healthy appe become another being. She who had always been disagr in bed or reclined in an arm-chair, was up the whole day, w and attempted to make herself useful about the room, ar parts of the house. In the first three months of her stay and before the employment of the pump, she had gained pounds in weight; two months afterward she had gained at least 16½ pounds, and in not quite six months, 24 to 25 pounds. For two years recovery has been complete, although the patient is by no means in easy circumstances."

The first communication regarding this *new method of treatment of gastric dilatation* was reported by Kussmaul at the first meeting of the Section of Internal Medicine of the Forty-first Congress of German Naturalists and Physicians in Frankfort-on-the-Main in September, 1867. Further observations and experience regarding this treatment, which, in the meantime, had become a method of gastric dilatation, were communicated by Kussmaul in his Freiburg pro-rector oration on the 9th of September, 1869, and in his classic treatise in the *Deutsches Archiv für Klinische Medicin*, vol. vi, 1869, which has become widely celebrated: "On a New Method of Treatment of Gastric Dilatation by means of the Stomach Pump."

Besides the new method of treatment, this epoch-making work of Kussmaul's furnished such an enormous amount of clinical material and so many new points of view for pathology, diagnosis and treatment that it became the foundation, and even a treasury, for most of the later investigations in gastric disease. Besides benign constriction of the pylorus from ulcers, cicatrices, hypertrophy, and malignant constrictions from cancer, here was found the complete clinical investigation of the simple dilatations of the stomach not dependent upon stenosis of the pylorus or of the duodenum, those due to *atony of the muscularis* in consequence of too great weight and extension of the stomach beyond its elasticity from polyphagia, or paralytic weakness in convalescence from exhausting diseases, for example, enteric fever, or which had been produced in nervous anemic conditions, as well as *paresis* of the gastric musculature in consequence of *fatty and choloid degeneration of the muscular fibers.* The definite *mechanical factors* were also considered which occasionally during life had caused symptoms of complete closure of the pylorus while in the cadaver a small finger could readily be passed through the narrowed pylorus into the duodenum. The *descent* in gastric dilatation of the *pyloric portion of the stomach* which is most bulged out and implicated, and the formation of the *fetal* (vertical) *position* of the stomach were also discussed, as well as the possibility of a reflex *tonic spasm of the hypertrophied pylorus,* which is manifest to a decided degree in consequence of the irritation of the sensory nerves of the pyloric region produced by the acid contents which cause increased peristalsis in this neighborhood.

That this increased irritation of the gastric mucous membrane from stagnating gastric contents produces reflexly an *increased,* even *continuous* secretion of fluid, and not only of HCl-containing gastric juice, was at that time unknown to Kussmaul. It did not, however, escape the sharp eye of the investigator that in his patient suffering from tonic muscular spasm the amounts of fluid ejected by vomiting and brought up with the stomach-pump were much greater than those introduced; the decidedly diminished amounts of urine in such patien believed for a long time, as I know from his personal comm a kind of transudation into the stomach, but he was unal exact clinical proof of such an influx of fluids; as is well k perimental success was achieved only a few years ago (1893) On the other hand, the significance of the marked, and occurring, losses of water in some cases of gastric dilatation to Kussmaul; he considered the tonic muscular spasms w described by him (so-called gastric tetany) to be the *consequ inspissation of the blood and a drying of the nerve and m* sequently, at that time, he laid great stress upon *the introd in the form of meat, broth, and wine enemata,* which were a intestine with beneficial effect. As an additional therapeutic we obtained simultaneously with the stomach-pump, we must maul's experience that the dilated stomach which is no lon retaining large amounts of fluid or food is still able to intestine *small portions given at stated intervals,* and, finall ment of the *hypogastric bandage* to prop up the descend stomach.

Kussmaul, in his classic work, refers at different points t of the stagnant contents of dilated stomachs in regard to a the appearance, the composition, and the degeneration of th in such a manner as to sound like a challenge to investig these questions. In truth, all of the clinical laboratories, Kussmaul's Clinic, were actively at work on this line, but w by the Franco-Prussian war and, later, by the great clini from Freiburg to Strassburg. The contents of diseased later, those also of healthy stomachs, were subjected to chemi and subsequently also to bacteriologic, investigations. Nor logic processes of digestion attained more and more promine a clinical and a professional standpoint. With an eagerness w the tube was introduced into the stomach and gastric conten up as if buried treasures were being restored to daylight.

Thanks to these eager investigators, we gradually becam importance in diagnosis and prognosis of *the presence or ab chloric acid,* not only in gastric dilatation but also in gastr general. *Simple color reactions* were found which, even showed the qualitative presence of hydrochloric acid and o while analytical methods by measure and weight were form numbers to determine quantitatively the gastric acids. In physiologic experiments in digestion to show the activity of the gastric juice, and their presence or absence, these quant also made it possible to test the function of secretion of the membrane and the digestive power of the gastric juice. We learned to recognize an *amylolytic and a proteolytic stage of gastric digestion, also the difference between combined and free hydrochloric acid.* Gradually a period dawned in which we believed that the activity of the stomach and the degree of its digestive disturbance could be definitely measured according to the amount of hydrochloric acid present, and the other functions of the stomach, especially its motor activity, were not sufficiently appreciated. A reaction took place. In the meantime, however, the number of methods for estimating free and combined hydrochloric acid became enormous, and the literature of this subject has become so extensive that it can scarcely be reviewed; the future will determine what portion of this is necessary, useful, and of permanent value.

But, to return to our theme of gastric lavage, we must first mention *the instrument* which Kussmaul used to empty and wash out the stomach.

Kussmaul first employed the stomach-pump of the American physician, Wyman, which, according to Bowditch's reports (1852), was also utilized in draining empyemata. Then, however, he had pumps manufactured by the instrument maker, Fischer, in Freiburg, after Wyman's drawings, and these were also made use of by Reich in Stuttgart who designated them as Fischer's stomach-pump, without any mention of Kussmaul's authorship.

v. Ziemssen later ascertained that the honor of inventing this very useful instrument belongs neither to Wyman nor to Bowditch, but to a German instrument maker by the name of Weiss, who lived in London in the second decade of the last century. Ziemssen therefore proposed in 1870 that "the instrument which, by Kussmaul's brilliant discovery, rapidly attained such great distinction in the treatment of gastric diseases, should hereafter be designated as *the Weiss stomach-pump.*" At first Kussmaul, after once emptying and cleansing a dilated stomach, only employed the gastric pump when new difficulties arose, such as burning in the stomach, acid eructation, pain, a tendency to vomit and similar symptoms.

In consequence, the *daily employment* of the stomach-pump, particularly in severe cases, became more and more obvious, and it was also ascertained that for *emptying and washing the dilated stomach, the time when the stomach was still empty, therefore, the early morning,* was the best time.

As long as the patients remained in the hospital, this operation could be easily and regularly carried out early in the morning upon an empty stomach. After discharge from the hospital, however, the patients are unfortunate, "for a busy physician cannot find time to use the pumps for such patients for weeks or even for many months with the desirable regularity." These reasons induced Kussmaul to *teach the patients themselves to introduce the stomach-tube, and to wash out their stomachs.* His patients who had learned to introduce the stomach-tube and the stomach-pump for themselves—as the clinical histories of the year 1869 show—left the Freiburg Clinic with these instruments in their possession. It is very interesting to note the use which a patient of Kussmaul's, by occupation, made of the stomach-pump as soon as indiges This patient by no means adhered to the directions which h in the Clinic, but, nevertheless, he improved steadily. If th were forbidden, pastry, bacon, poor beer, or others, caused culty, he used the pump and washed them out—often twice d

“ In comparison to this refined behavior of the Black Fores how insignificant,” says Kussmaul (l. c., page 498) “is th *suetudo vomitandi* of Vitellius and other celebrated gourma Rome, who had at command only the maneuver of the palate- or the feather.”

But, from the onset, Kussmaul was not content with the tion of the stagnating gastric contents. On the contrary he employed the stomach-pump for irrigating the diseased g membrane with suitable fluids, designed to have a curativ limit processes of decomposition and the proliferation of fu

First, natural *alkaline waters* were employed, Vichy an however, were gradually replaced by corresponding artificial For the latter purpose, *borax solutions* (4–400), *phenyl acid* creosote water to 400 of water), solution of *sodium hyposu* grams to 400 of water), which, it was expected, would cause of disinfecting sulphurous acid in the stomach, also lign. raspat., of which 30 grams were covered with 400 grams and, after macerating for twelve hours, the colature was e action of stomach washing was assisted by *dietetic* treatmen prised the ingestion of small but frequent meals consisting could be easily digested and contained nothing that might pro acids; also the employment of soda-containing mineral waters given in amounts of a ½ schoppen, one-half to one hour befo before the first meal, for the purpose of dissolving the tough had formed in surplus, to neutralize the *abnormal acid,* an the secretion of normal gastric juice; finally, keeping the b means of sour milk, buttermilk, simple warm water enemata

The great enthusiasm aroused by the first communicatio before the Frankfort Naturalist Congress, and, even mor recognition in all professional circles of the world of the cess of his method of treatment, when compared with ot detailed explicitly in the *Deutsches Archiv für Klinische M* the rapid introduction of the stomach-pump into hospital an tice. With the publication of Kussmaul's method of treatme literature of the subject rapidly appeared which at first broug in regard to technic, various processes being described which non-employment of the stomach-pump, as this could not alw obtained.

In Kiel, Dr. Schorer, the assistant of Bartels, in the absence of a Wyman pump used the ordinary laboratory air-pump. Jürgensen, at that time professor in Kiel, inserted between pump and stomach a two-necked Woulff's bottle, in order to meet the objection that all fluids discharged from the stomach passed through the metal boot, and thus corroded and damaged the pump which was very difficult to clean.

Jürgensen, however, soon noticed that the stomach-pump was only necessary until the tube was filled which led from the stomach to the low-lying Woulff's bottle: *From that time on the indications for emptying the stomach by the action of a lever were given.* Jürgensen obtained the latter just as well by abdominal pressure as by the suction pump. *For this reason the use of the stomach-pump was discontinued, the stomach was no longer pumped out but evacuated.*

In his article: "An Addition to the Local Treatment of Gastric Diseases" (in the *Deutsches Archiv für Klinische Medicin,* vii, 1870), Jürgensen describes the *simplest method* embodying the lever principle, which he advises for the busy practitioner as the most serviceable, in the following:

"By the aid of the tube which is introduced into the stomach the latter, if empty, is filled with a fluid to be utilized in washing; naturally, if the stomach contains sufficient fluid this is unnecessary; then by means of a glass tube or a hard rubber and glass tube (1.5–2 meters long, and one centimeter in diameter) an ordinary rubber tube is attached to a sound.

"*The patient now stands upon a chair, and either coughs or retches.* The flow from the stomach immediately begins. The emptiness of the stomach is indicated by a sudden jerking of the rubber tube; and the stream of outflowing water at once becomes thinner.

"Thus, in a brief time, a large amount of fluid may be forced through the stomach. To be certain that my lever may actually be filled without difficulty by the prelum abdominale, I fill the stomach with water, and pay no attention to the slight inconvenience which the patient appears to experience." (Jürgensen.)

Compared with gastric lavage as practised to-day, this method described by Jürgensen as the simplest appears to require considerable assistance from the patient who must get upon a chair with a sound in his stomach. Nevertheless, we owe it to Jürgensen that the pump was proven to be unnecessary.

Jürgensen did us the further service of attempting to substitute *soft rubber sounds* for the stiff French and English stomach sounds which were then in use.

These stomach sounds were of very small caliber, and had upon their anterior end two or four small fenestra, in consequence of which they very readily became clogged with the remains of food and even with mucus. A very disagreeable feature for the patient was that, as soon as clogging occurred, the sound was taken from the stomach, cleansed, and reintroduced. This process was improved by Jürgensen who obtained from the

factory of Galante in Paris an excellent tube having a le
the walls being 2 mm. in thickness, and 10 mm. in dia
tied with a silk thread over an acorn-shaped ivory button
such a manner that the tube nowhere extended above the le

In the base of the lumen of the tube the ivory button
tion 3 mm. deep, which was intended to receive the an
whalebone sound acting as a director to prevent its lateral

Laterally, 2–2½ cm. above the ivory button, a hole was
corresponding to its lumen. Jürgensen preferred *one* ope
chiefly because of the durability of the tube. If he po
one opening of the sound this flowed from it in a full st
sound itself, owing to the well-known phenomenon of "
move with great rapidity in the direction opposite to the o
lateral deviation of the tube, Jürgensen believed, especiall
sound on its longitudinal axis, that he was able to *douche*
the instreaming water.

When it was desirable to prevent the lateral motion
the stomach, a second small opening, opposite the outlet,
this purpose.

The tube-sound was so prepared that the stylet was at
into the hollow orifice of the acorn-like attachment, the
drawn over it and stretched, and, finally, a clamp was att
of the tube opposite the acorn, over the tube and the directi

"When the sound equipped in this manner has bee
5 cm. into the esophagus, the clamp is removed, the whale
drin) may be withdrawn, and the soft tube is passed down
and held so that it cannot be forced up by the retching" (

A further simplification of the technic of gastric lava
tion, by Rosenthal, of a T-shaped tube with a cock int
which, being placed in different positions, might be con
vessel containing the water, with the descending outfl
stomach-tube with the outflow tube, or the vessel contai
the stomach-tube. A similar technic was recommended b

Then experiments were made with *double stomach sou*
Auerbach in the Clinic at Kiel, then by Ploss, Hodgen, Ap
Hodgen (St. Louis) invented a simple hard rubber pu
ball valves were so arranged that the mere turning of th
trary direction was sufficient to reverse the stream. Hodge
the method of emptying the stomach by connecting a rub
gastric sound, and filling it with water before introducin
the stomach.

According to whether the vessel containing the end o
is higher or lower than the stomach, fluid may be allowed
stomach or may be withdrawn from it.

Extreme simplicity seemed to be attained in the apparatus employed by Th. Biedert; it consisted of a stomach-tube, a glass tube 6 to 8 cm. long, which was inserted into the former, and which at its other extremity was connected with a long rubber tube, and, finally, with a funnel into which the water could be poured (1873). To permit the water to flow from the stomach, Jürgensen, as mentioned, still believed it necessary that the patient with the full, or even distended, stomach get up on a chair. In 1875, Holland Cotton thought it sufficient for the patient to stand during the lavage; Biedert filled and emptied the stomach by raising and lowering the funnel.

A fortunate advance in the technic of gastric lavage was the introduction of *soft stomach-tubes* by Jürgensen. These instruments had the disadvantage that their flexibility was soon lost, from the fact that a mandrin of whalebone or rattan was introduced into them to stiffen them until the larynx was passed. It is greatly to Ewald's credit to have shown (1874) in a case of nitrobenzol poisoning that even a *very soft* rubber tube, or an ordinary smooth gas tube, may be introduced into the stomach and utilized for gastric lavage. In a discussion of Oser's communication ("Die Magenspülung mittels des elastischen Schlauches," *Wiener med. Presse,* 1877, 1), Nothnagel, however, emphasizes (in Virchow-Hirsch's Jahresberichten) that he also, without any knowledge of Ewald's publication, employed in his Clinic for some years a smooth rubber tube with a funnel for simple gastric lavage.

Ewald's gas tubing was the prototype of the improved tube which later came into use,—stomach-tubes which were open below, i. e., with one central opening only. Jürgensen's ivory knob at the end of a soft tube fell into disuse when, with the technical improvement in the rubber industry, closed, rounded tubes, with one or two lateral openings, stomach-tubes patterned after the Nelaton catheter, were then manufactured. Such tubes were first employed by Leube; by and by, however, they came into general use, and have been exclusively employed in gastric lavage.

The technic of the introduction of the sound in gastric lavage which was carefully constructed and practised in Kussmaul's Clinic, had then to e learned and practised by others who desired to utilize this new method f treatment. It goes without saying that in such mostly autodidactic andling of the sound the patients must have been obliged to bear a great eal. I do not believe I am far wrong in assuming that the widely prevalent ar of the stomach-pump which still exists in the minds of many patients riginated in that period when physicians were learning to use the stomach und and the stomach-tube.

Recognizing these evils, v. Ziemssen was the first to advise care in the nployment of the stomach-pump (1872). Then Biermer (1874) pubhed a communication concerning inflammation of the esophagus and

of the peri-esophageal connective tissue following injuries
This author was later followed by Leube and others. In
of the stomach sound, as well as also in the use of the sto
in withdrawal of the sound from the stomach, now an
occurred which, under some circumstances, made these pr
ous. It repeatedly happened that portions of the mucous
torn off by the sound, sometimes fragments from 1 to 3 cm.
mm. in breadth, therefore presenting quite a surface. I
cases of injury to the gastric mucous membrane were seri
noted; now and then more or less decided hemorrhage and s
but recovery was rapid and so complete that even in thos
there was an opportunity later to hold a necropsy no visibl
found in the stomach. Besides these lacerations in the m
of the stomach, there are reports in literature of injuries t
and esopheageal mucous membranes, perforations of aortic
orrhages from esophageal varices, of erosions and superfici
the gastric mucous membrane. Moreover, the mucous me
perforated by using hard sounds, and the overfilling of t
fluid has caused decided ruptures of continuity in the
membrane.

The number of accidents of this kind noted in literatu
small when we consider the extensive and world-wide use
tube. However, in the opinion of a celebrated author (I
not exactly represent actual conditions. For instance, t
of particles of the gastric mucous membrane with a stoma
now said to be much more frequent than might be as
publications.

In the year 1872 Ziemssen also advised us to be caref
certain sounds, particularly the black ones (French), and
friable borders at their openings. Emminghaus did the sa
pared the action of a sharp opening in the sound with
chisel, and he believed the central opening in the sound
as had previously been stated by Emminghaus. Ziems
minute directions for preventing injuries with the soun
Measure the distance between the epigastrium and the tee
before its introduction, and mark upon the latter a line so
ment will not be forced so deeply into the stomach as to da
curvature; 2. Never begin to pump out the stomach before
ing warm water (at least half a liter), unless a short time
amounts of fluid have been ingested; otherwise, the gastr
brane may be drawn by suction into the opening of the t
torn away.

With the general introduction into practice of the s
these dangers have been almost obviated. Gastric lava

regarded as a harmless procedure provided the cases are suitable for lavage and the instruments correctly chosen, and that we adhere to the technical rules primarily given by Kussmaul, which are still to be explicitly discussed. The greater certainty which has resulted from this manner of handling the stomach-tube no longer limits the employment of lavage to the *dilated* stomach. Kussmaul advised the employment of lavage *as early as possible* in reflex spasm, in stenosis of the pylorus, and in pathologic conditions leading to degeneration of the gastric walls. In consequence of this Reich in Stuttgart (1869 and 1870) and Schliep in London treated chronic diseases of the stomach, catarrh, ulcers, and digestive disturbances from other causes with the stomach-pump, and also employed the remedies advised by Kussmaul in irrigating the affected gastric mucous membrane,—solutions of sodium bicarbonate, potassium permanganate (1 to 100, of this 2 to 4 ounces in a vessel filled with water), carbolic acid, chloralum (an English preparation), boric acid and tincture of myrrh. In an article from Kussmaul's Clinic, "On the Treatment of Gastralgia with the Internal Stomach Douche, and Remarks Regarding the Technic of Sounding the Stomach," Malbranc (1878) described the anodyne effect of sprinkling the gastric wall with warm water, and particularly with warm carbonated water (100.4° F.). Rosenheim, in 1892, advised a particular sound with many small lateral openings for douching the stomach. In the same year Löwenthal suggested in the treatment of hyperchlorhydria and gastralgia spraying the gastric mucous membrane with solutions of silver nitrate (1 to 1000) and Einhorn employed the spray in various gastric affections.

The communications of Cahn, from Kussmaul's Clinic, upon the treatment of intestinal obstruction by gastric lavage (1884), received merited attention. In many cases of ileus, the results of repeated, even of a single, irrigation of the stomach, as I later had abundant opportunity of convincing myself, were remarkable. The benefit was brought about by the removal of gas and fecal masses lodged above the stenosed area. This removes tension and the limitation of space. Decided peristalsis and vomiting cease, and food may then be introduced.

I must also attribute to Kussmaul's initiative the method, which I have systematically described and practised, of covering with bismuth the ulcerated and bleeding surfaces in the stomach, these having first been cleansed (1893).

The beneficial effect of gastric lavage upon the bowels in dilatation of the stomach, Kussmaul mentioned in his article to which we have so often referred. This was noted in all cases in which gastric lavage was at all serviceable, and he looked upon it as a suspicious sign if, after prolonged gastric lavage, amelioration took place, and the gastric symptoms improved, but constipation stubbornly persisted.

In conjoined labor with the venerable master for almost ten years, *I have availed myself of the benefits of gastric lavage in intestinal disturb-*

ance to an enormous extent, not only when constipation ex more frequently when prolonged diarrheas were present whic their origin in disturbances of gastric function and organic stomach. Here I cannot refrain from mentioning the fav gastric lavage in many diseases of the liver, in diseases of t combined with jaundice, in chlorosis and anemia, in mar of metabolism, and in intoxications. Gastric lavage has n effect, but, in those cases where the pylorus opens during general effect in that the water which streams into the sm not regurgitated into the stomach, and not expelled externa blood and the liver, washes out the blood and the vessels, anc cleanses and washes out the kidneys just as effectually as tl spring cures.

Furthermore, I must call attention to the value of ga diagnosis; this was evident from the beginning and has be apparent in the course of time. "We first empty the stoı the palpating finger may detect *tumors* which cannot be : the stomach is filled. Secondly, lavage is a very valuable ı to differentiate dilatation of the stomach from *dilatation oj colon;* this was formerly very difficult, so much so that, a my own experience, the greatest clinicians occasionally n takes" (Kussmaul, 1869).

Finally, gastric lavage enables us to arrive at conclusion motor condition of the stomach, and Leube did us a las utilizing gastric lavage to estimate the time necessary for g and for the *diagnostic determination* of the motility of th systematic washing out of the stomach a certain time afte of various foods and drinks Leube, and, following him, F absolute knowledge concerning the activity of the stomach, digestibility of food. The results obtained became the ba dietetics. This was the origin of the well-known diet sch as well as the tables of Penzoldt, which are so useful in pı the gastric digestibility of ordinary food and drink.

The motor function and the activity of the stomach by Leube as sufficiently good if, in the course of from five after a test-meal at midday consisting of soup, meat and a washing of the stomach showed it to be almost or entirely this time, considerable quantities of the midday meal are the stomach, digestion is slow or imperfect. Such a stoma is taught by clinical experience, will during the night rid i tents and become empty by the morning even if another eaten in the evening before complete emptying. I, ther stomach which does not become empty during the night, and in the morning remains of food from the previous day a as deficient in a motor respect only. A flaccid or atonic stomach is occasionally not empty early in the morning on account of an error in diet, either by overloading and over-distending it, or because of improper food and an improper mode of life, while it may be quite capable of digesting and assimilating suitable food. On the other hand a dilated stomach in consequence of moderate stenosis of the pylorus may, for a long time—until improved by washing—show motor insufficiency, but in severe cases it is permanently in this condition, in which case it is never empty early in the morning, and is therefore incurable by gastric lavage.

At this point I shall mention what Kussmaul found to be the limitations of his method in the treatment of gastric dilatation. He was able to *cure dilatation of the stomach* by gastric lavage when no, or only moderate, constriction of the pylorus or duodenum was present. *No cure, but amelioration only,* could be brought about by gastric lavage in the following conditions:

1. In *malignant stenosis of the pylorus,*
2. In *very-decided cicatricial narrowing of the pylorus,* and
3. In *moderate narrowing, when the gastric wall, in consequence of chronic gastritis, had suffered such extreme degeneration as no longer to be capable of retrogression.*

For the cases which at that time (1869) were looked upon as incurable, gastric lavage was evidently a remedy that, *if employed early,* would decidedly prolong life and markedly improve the nutrition.

"Naturally, a cicatricial narrowing which cannot be dilated even to the extent that a goose quill may pass through the pylorus, can never be cured by gastric lavage." With keen discernment, the great master continues: "*Whether in the most daring ages of a distant future, an attempt may be made to produce radical results by gastrostomy, and to form a gastric fistula and dilatation of the stricture by the knife or sound, no one can to-day positively assert.* We fear that even the proposal of such a method of relief may expose us to silent or expressed ridicule." Kussmaul, *Deutsch. Archiv für Klin. Medicin,* vi, p. 485.

Perhaps it was the inspiration of this ingenious thought of Kussmaul's which stimulated his friend Billroth (who was, unfortunately, too early removed from his sphere of activity) to attempt the cure of heretofore incurable gastric diseases, in which internal treatment had been of no avail, by operative procedures.

But this was by no means left to a later race of surgeons; by means of antisepsis and asepsis the contemporaries of Kussmaul have been enabled to operate on diseased organs within the cavities of the body. Since that time surgery of the abdominal organs—and, by no means least, gastric surgery—has had a brilliant and wonderful success. At the turn of the century the realms of internal medicine and surgery are no longer, as a few decades ago, sharply, almost diametrically, opposed to each other, but, in

united labor, such as Kussmaul saw with his mind's eye or
physician and the surgeon are working hand in hand to
high goal: To cure!

THE CLINICAL EMPLOYMENT OF GASTRIC L

Owing to the results achieved by gastric lavage and diet,
of diseases of the stomach has become one of the most gr
the internal clinician. In the interest of the practising
to be greatly deplored that this realm, which is consta
threatens to become the domain of specialists. For the p
satisfaction which springs from the consciousness of havi
sick from distress, and which richly compensates the phys
weary hours of his professional life, can scarcely be greater a
version or forceps extraction, after the curative removal of
cealed abscess, after the removal of an obstruction to res
aspiration of a pleural exudate or of ascites, or after th
relief of an over-distended bladder difficult to reach—than
lavage, which frees from his painful condition an unf
stomach has no longer the power to empty itself.

The dread of disagreeable consequences which the first
lavage brings to the physician as well as to the patient
possible danger of certain unfortunate errors in technic fo
responsible, may restrain some physician, whose position i
patient is often very difficult, from employing this otherw
method. Nevertheless, particularly in severe disease, the
lavage carried out at the right time is so great, and th
ensues from neglecting the necessary emptying of the st
so considerable, that personal considerations must be set
with the methods in vogue to-day it is quite possible to u
tube for diagnostic and therapeutic purposes in such a
to the patient may be absolutely avoided.

The technic of gastric lavage has become of such vast
tance, for the physician as well as for the patient, that I am
detail some *old, well-tried rules* which I learned and con
association with my highly honored teacher, Kussmaul.

The stomach-tube, as is well known, serves *for diagnostic*
purposes. We test the function of the stomach to aid us
of a gastric affection or in the correct decision as to the
stomach performs in a complex of pathologic phenomena.
prises two parts: A test of the gastric contents at the heig
and a test lavage.

A test of the gastric contents is made a certain time a
istration of a test-meal, usually from three to three and a

a meal consisting of gelatinous soups (250), roast beef (200), and mashed potatoes (200), or one hour after a trial breakfast consisting of tea and a roll. By means of the stomach-tube a sufficient quantity of the gastric contents is obtained for chemical, physiologic and microscopic investigation, so that we may examine the *secretory* and *fermentative* processes in the stomach.

The *trial lavage* of the stomach takes place early in the morning, the stomach of the patient being empty, and enables us to form conclusions regarding its *motor* activity, as well as also in regard to many pathologic conditions, such as catarrh, secretory irritative phenomena, continuous secretion of gastric juice, and stagnation and decomposition of food.

In the trial lavage of the stomach, which naturally should be empty, we must not neglect to secure separately the stagnating contents or the first fluid which comes from the stomach, and these should be examined chemically and microscopically—at least, with *litmus paper* and with *Congo paper*. If circumstances permit, a test of the gastric contents should first be made, and then the trial lavage should be attempted; there should be a day of rest between these procedures.

How these results assist in the diagnosis and indications for treatment will be described later.

The *therapeutic indications* obtained by the aid of the gastric tube are manifold.

Frequently the stomach-tube is used for the purpose of emptying and washing the stomach. Such stomach washings are indicated in all cases in which the stomach, early in the morning, is not empty, but in which are found remains of food from the preceding day (or even earlier), mucus or gastric juice containing hydrochloric acid such as occurs in chronic gastric catarrh, and a continuous flow of gastric juice in consequence of permanent irritative conditions of the gastric mucous membrane.

Even if the stomach early in the morning is quite empty, gastric lavage is often performed. Washing or spraying the gastric wall with the stomach-douche may be designated as the best remedy to stimulate the appetite, the flaccid gastric musculature and the secretory glandular apparatus, and also to relieve irritative conditions and pain or vomiting. All of the symptoms just mentioned are indications for the employment of the stomach-douche. Instead of water of varying temperature (usually from 86° to 95° F.) saline solution, mineral water and diluted drugs of various kinds are employed, according to circumstances.

Gastric lavage is also frequently and successfully employed in disturbances of function and in diseases of the intestine and of the liver, in conditions of abnormal blood-mixture, in renal diseases, and in acute poisoning, as well as in chronic states of intoxication, for example, uremia.

The indication for gastric lavage in ileus (intestinal obstruction) deserves special mention.

Before each introduction of the stomach-tube or of the sou must be carefully examined, since this alone makes it possib inate in the choice of patients, and to exclude those in whom might be dangerous.

In deciding what cases are suitable for gastric lavage it m bered that the first introduction of the stomach-tube alm produces excitement, retching, and nausea, sometimes als respiration and cardiac palpitation. Some patients, in c psychic stimulation, hold the breath, become cyanotic, and, exertion of the prelum abdominale, bring about a decided pressure. In irritable and anxious patients, all these sym more rapidly and reach a higher degree than in persons temperament who are able to control themselves. The of those who suffer from diseases of the respiratory a organs.

For this reason persons who, on account of diseases of t passages, the lungs or the pleura, can only complete their res change of gases with difficulty, are markedly disturbed by and, under some circumstances, to them it even becomes da effect is similar in those who have a disease of the heart, who is weakened or degenerated, or whose valvular disease is compensated for as to be unable to withstand great variat pressure. *Therefore, we refrain from stomach washing an evacuation to promote the diagnostic investigation of the sto when severe pulmonary and cardiac affections are present.* the conditions are such as to necessitate the introduction of and if it is believed that the value of this procedure will possible danger from its use, the expert, relying upon the acc of the stomach-tube while carefully regarding the rules of th nevertheless, attempt to introduce the instrument. If th successful, with like caution others may follow, for a sensibl becomes accustomed to its use. Many a phthisical patient b out of the stomach has gained an appetite, acquired new prolongation of life. I even believe that *incipient phthisis* sons may be arrested with the removal of the anorexia by and subsequent plentiful feeding, and that in this way a brought about.

As a rule, we avoid the use of the stomach-tube if we suspect that the walls of the vessels in any part of the bod to withstand a rise in blood-pressure brought about by the i the sound, which is apt to produce retching, vomiting, or of the abdominal press. *Preceding hemorrhages* (hemoptysi melena, apoplexy) *or a tendency to hemorrhage still present. tous arteries, valvular disease of the heart demonstrable by* *auscultation, and even suspected aneurysmal dilatation of the aorta,* are valuable guides.

Recent and profuse hemorrhages from the stomach or from the lungs, high-graded atheroma, severe cardiac disease, and aortic aneurysm are always contraindications to the employment of the stomach-tube.

Capillary hemorrhages from the inflamed, perhaps also eroded, gastric mucous membrane, and parenchymatous bleeding from ulcerated, decomposing growths in the gastric wall, may, under some circumstances, also be contraindications; usually, however, they are not; but such hemorrhages generally cease if, after the discharge of accumulated and decomposed masses, the stomach is again able to contract. In hemorrhages of any kind, even during the menstrual period, I only very exceptionally make a gastric examination with the stomach-tube and perform gastric lavage. During menstruation the gastric mucous membrane of many women is somewhat inclined to hemorrhage. For this reason I interrupt the treatment by gastric lavage, and resume it only two or three days after the menstrual period. According to experience, *women during menstruation digest food less well, and they secrete a weaker gastric juice* than at other times, therefore it is well to defer the investigation of the gastric contents.

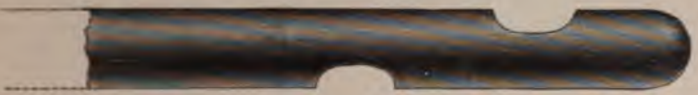


FIG. 2.—Terminal piece of a soft tube as found in the instruments usually on the market.

To obtain the gastric contents and for gastric lavage we employ exclusively *soft stomach-tubes,* and preferably those of English manufacture (Jaques's esophagus tube) on account of their smooth surface and great flexibility. However, these English tubes have glaring defects which for some unaccountable reason the manufacturers do not remedy. *Their openings are frequently not large enough, and the margins of the openings, without exception, are so sharp that they readily injure the mucous membrane, particularly the mucous membrane of the stomach,* as may be seen from Figures 2, 3, and 4, which show the configuration of the tubes in natural size. This defect may be remedied by burning out the openings, and rounding off the edges with a hot darning needle or a hot glass rod, as may be seen in Figures 5 and 6. But, after this treatment of the margins of

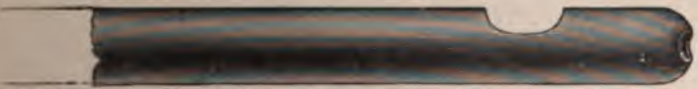


FIG. 3.—Terminal piece of a soft tube with a lateral opening, and a central opening in the longitudinal axis of the tube. This tube appears to me to be most dangerous to the mucous membrane of the pharynx, of the esophagus, and of the stomach.

the openings, the rubber is rough for a time, and must be with pumice stone. Besides, the tube has for days an odor ber, and, sooner or later, small rents appear in the edges wh burnt and soon render the tube unfit for use. It is to be h

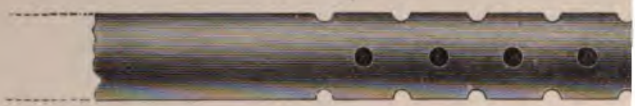
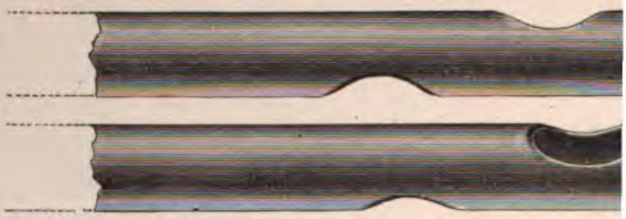


FIG. 4.—Terminal piece of a soft tube with a central and numerous late said to be more suitable for irrigation of the stomach than tubes wi lateral openings.

manufacturers will speedily place on the market more ser with rounded, polished edges that will meet practical requir

The introduction of a staff or mandrin into a soft st decidedly objectionable, for, with a mandrin inside, the s becomes a hard sound with all of its defects and dangers. required in gastric lavage and artificial nutrition when it overcome an obstruction in the front of the stomach, in na esophagus, or in deep-seated dilatations of the esophagus, f some ante-stomachs.

Preparation of the Patient.—Before the first introd stomach-tube I have never failed minutely to inform patie all intelligent regarding the purpose and utility of the pro their attention to its harmlessness, and to teach them how to selves during the operation. Whenever possible, I have



FIGS. 5 and 6.—Terminal piece of soft tubes whose openings have been hot glass rod and thereby rendered harmless.

nervous patients an opportunity to see how willingly othe practice swallow the tube themselves and permit lavage.

The patient now sits erect in a chair, or, if in bed, sits of the bed. A basin is given him in which to catch the sal from the mouth, or the vomited material, and which he h hands, so as not to disturb the physician by making motions If dealing with patients who have very sensitive pharyngea branes, such as smokers who retch easily and show a ten the introduction should be preceded by gargling with a watery solution of potassium bromid, or by painting the pharynx with cocain, when the sensitiveness and reflex irritability of the structures of the pharynx may be so controlled that we may proceed with the introduction of the tube. Artificial teeth must, naturally, be removed. The patient should be allowed to drink some water before the lavage, so that the mucous membrane of the mouth, tongue, palate, pharynx and esophagus may be moistened and more readily permit the passage of the tube. Where there is a very irritable stomach and a great tendency to vomit, the patient should even be made to swallow a considerable quantity of water before the introduction of the tube, so that this will not reach an entirely empty stomach and, by direct contact with its walls, produce great irritation.

Naturally, if we desire to obtain the gastric contents for the purpose of analytical examination, water is not permitted before the introduction of the tube. In this case the prevention of retching and a tendency to vomit are not at all necessary for, under these circumstances, the evacuation of the gastric contents is only facilitated. In this latter operation —in case it is done without a pump or aspiration apparatus—injury to the gastric wall is unlikely, since the stomach is not empty, and the tube can hardly come in contact with the walls of the stomach.

Preparation of the Stomach-Tube.—According to Luschka's plates, the ninth spinous process of the thoracic vertebræ corresponds exactly to the position of the cardia, provided the location of the abdominal organs is normal. Before the first introduction of the tube we count on the patient's back the spinous processes of the vertebral column up to this point, to which we apply the upper opening of the tube, and measure along the vertebral column, passing alongside the ear to the incisor teeth, and mark this point upon the tube with a waxed silk thread which we tie around the tube, or with a colored pencil. If the tube is now introduced up to this point it is certainly in the stomach, provided this organ is in its normal situation, and it is unnecessary to shove the tube up and down to ascertain whether it is in its proper position, as this will cause retching and a tendency to vomit. Before the introduction is begun the end of the stomach-tube is moistened in luke-warm water.

While the patient sits erect in a comfortable position, with the head raised, the mouth open wide, and the point of the tongue placed against the lower incisor teeth, the rounded end of the stomach-tube is pushed over the dorsum of the tongue up to the posterior pharyngeal wall. The moment the tube reaches this point, the patient slowly bends his head forward and simultaneously attempts to swallow. The sound glides along the posterior wall of the pharynx, along the vertebral column to the cricoid cartilage, often even beyond this. Now we pause for a moment, ask the patient to breathe deeply and regularly, and then to swallow again, when the tube will slide downward behind the larynx, and with a slight push on the part of the physician the instrument is rapidly int the required mark. When this is accomplished the tube is hel the patient breathes quietly and regularly, and, in the mea of hard rubber which encircles the tube, is put between his te this ring he may bite.

The further procedure depends upon the purpose for w has been introduced.

To Test the Gastric Contents.—If it is desirable to obtai gastric contents for a test of the chemism of the stomach, patient, after the tube has been introduced and is in the rig contract his abdominal muscles as if he were having an ev this exertion of the abdominal press, the gastric contents a through the tube and may be caught in a vessel held for the

It may happen that, on pressing, only air comes up thr and none of the ingesta. The tube is then gently pushed and after a few deep respirations renewed attempts at expres With very flabby abdominal walls, the result may sometim about by pressing a hand upon the back and one upon th assist the abdominal press, or by raising the protruding a to bring the descending stomach and its contents nearer t the tube.

If these manipulations are futile, either the stomach is tube is clogged by insufficiently masticated particles of mea meal which have not yet been dissolved in the stomach and to pass. It is not always easy to determine which of these tw is the case. If, on repeated attempts at pressing, only through the tube—and this may be recognized by the sound panies it—this is in favor of the *stomach being empty.* If hand, upon attempts at pressure nothing or only the sound o which suddenly ceases, or if the stream of stomach contents flowing from the tube and does not again flow when pressur *tube is clogged.* It may be that the tube is not clogged b food, but that *a fold of the gastric mucous membrane is s opening of the tube* and thus closes the opening. The lat must be borne in mind if, upon pressure, neither air nor sto come forth.

In such cases there is danger, by rapid displacement o injuring the gastric mucous membrane, or of tearing off mucous membrane which has been sucked into the opening Great care is necessary in moving or in withdrawing the assured of the results when gastric contents are not brough the tube, hold it firmly, connect it with the other parts apparatus, and permit a measured quantity of lukewarm wat the stomach. If this water, upon lowering the funnel, reg pure, the stomach has passed the test-meal into the intestine, and the tube may now be removed with the observance of the necessary precautions. The attempt to obtain the gastric contents has, under such circumstances, failed, and if we desire to obtain further insight into the chemism of the stomach the test-meal must be repeated at some other time, and at an earlier hour. At all events, the operation gives proof of a normal, even increased, motor activity (hypermotility) which compensates for secretory disturbances.

A proof that the tube has been clogged, and that the stomach is not empty, is the fact that water poured into the stomach reappears mingled with gastric contents when the funnel is lowered. Qualitative investigation for hydrochloric acid, at least with Congo paper, may be made in the fluid thus obtained, but no *quantitative results* are furnished. Under these circumstances, therefore, another test-meal must be given, the attempt be repeated another day, with due care that the meat has been finely chopped so as to avoid clogging of the stomach-tube. In the first attempt, I do not have the meat for the test-meal chopped, for it often aids us in the etiology and treatment of many digestive disturbances to know how the patient chews.

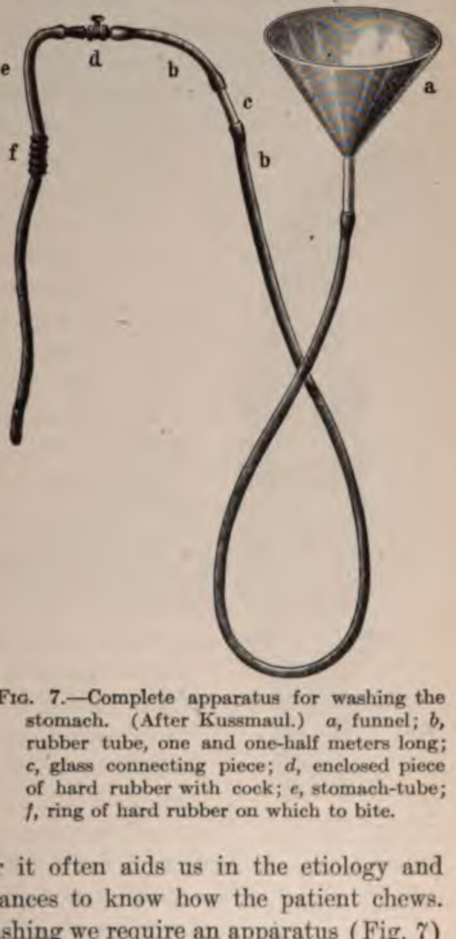


FIG. 7.—Complete apparatus for washing the stomach. (After Kussmaul.) *a*, funnel; *b*, rubber tube, one and one-half meters long; *c*, glass connecting piece; *d*, enclosed piece of hard rubber with cock; *e*, stomach-tube; *f*, ring of hard rubber on which to bite.

Gastric Lavage.—For stomach washing we require an apparatus (Fig. 7) which is composed of the already mentioned stomach-tube and the ring which is placed over it and held between the incisor teeth (Fig. 8) for biting, a glass funnel holding from one-half to three-quarters of a liter, and a rubber tube one and one-half meters long interrupted in its lower third by a short glass tube which is connected with the funnel, also an inserted piece by which the rubber tube is readily attached to the stomach-tube. This enclosed piece (Fig. 9) is made of hard rubber, and is of the form and size depicted in the illustration. The round end, *a*, is placed toward the funnel and attached to the rubber tube. At *b* there is a faucet which is usually firm and air-tight, but, under certain conditions which we

will learn to recognize, may be removed for a short time.
narrowing end, *c*, of the inserted piece answers the purpose
the tube with the stomach-tube and, on account of its shape,
any caliber.

If the stomach-tube has been introduced according to
given, the biting ring is between the incisor teeth, and ever
position, we fill the lowered funnel with lukewarm water, a
it so that the water flows into the stomach. When proper
given I permit the funnel to be fill
the tube has passed the larynx and
introduced and elevated.

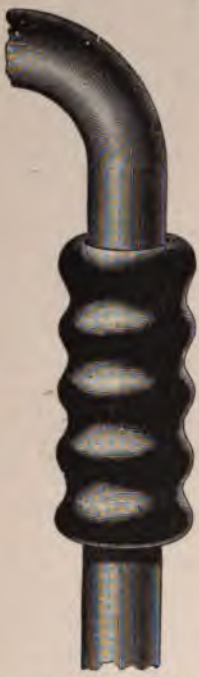


FIG. 8.—Ring on which to bite (of hard rubber) drawn over the sound, and readily moved about upon it.

The rapidity with which the water
stomach depends upon the pressure o
of water, i. e., upon the elevation of th
the degree of contraction of the gas
ture, and upon the pressure of the pr
nale. Under some circumstances, as i
coughing, nothing flows into the stom
when the funnel is raised very high ei
for washing or some of the gastric co
through the tube. On the other hand
ach walls are flaccid, on deep inspi
consequence of the diminished intra-al
sure the water is aspirated into the
actually streams into this organ. It m
if the funnel is raised high, that a bul
by means of this, simultaneously with
is aspirated into the stomach and ca
distention of the organ. A simple ar
slanting the funnel, may prevent this

Before the funnel becomes quite em
rapidly lowered so that no air can pass
and the continuous flow of water thro
not interrupted. On lowering the fu
action takes place, and the water regurgitates into the funnel
be collected, examined, its reaction tested with litmus or Co
we should carefully observe whether as much water flows out
as has been poured in. By this precaution, it is quite imposs
the stomach by over-filling it with fluid. In the stomach-t
wash-tube, even after emptying the funnel, a column of flui
glance at the glass tube within the stomach-tube will show of
column consists. If it is clear, or but slightly turbid, we s
the lavage. The lowered funnel is refilled with water, is r
ered again, the water used for washing is collected and emp

If, however, the fluid in the tube is quite turbid, or decidedly mixed with mucus, remains of food or the like, the faucet at the inserted piece is removed for a moment so that air enters the tube and the contents flow toward the funnel. When this is done, the faucet is reintroduced, the washing is continued, the faucet is again removed, and if the water returned a second or third time through the funnel is not yet clear, this process is continued until the water flows clear. Without the enclosed piece with the faucet—which, by the way, is the invention of an artillery officer who was treated in Kussmaul's Clinic for dilatation of the stomach—the soiled water is again forced through the tube and returns to the stomach. The process is then unnecessarily prolonged, and this is an evil of some consequence in debilitated patients, one which is obviated by the introduction of the enclosed piece, which is as simple as it is ingenious.

The stomach, as a rule, is washed until it is clean, provided the patient is able to bear it. In very irritable patients we must reckon upon their resistance, and, particularly in the first lavage, we may be obliged to desist before this end is attained. Even with patients whose activity and self-control is only slightly diminished, days frequently pass before the remains of food from former meals can be removed from a dilated stomach.

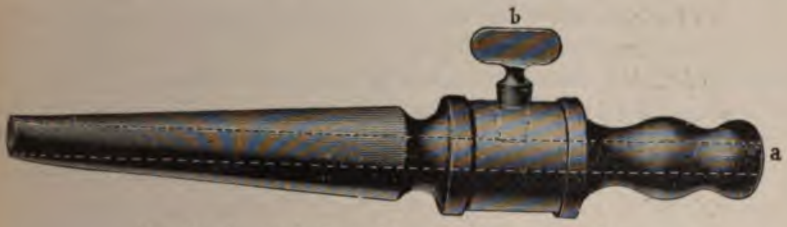


FIG. 9.—Inserted piece of hard rubber with cock.

Besides, a return flow of clear water does not always indicate that the stomach is actually clean. For example, *mucus,* which often thickly coats the gastric walls, *dissolves but slightly or not at all in pure water;* the water may, therefore, flow off clear while a mucus coat, rich in microbes and ferments, may still adhere to the gastric mucous membrane. If, in such cases, alkaline waters or solutions of soda and table salt are used for washing, it is frequently astonishing to see the amounts of mucus brought up.

The conditions are somewhat different in a flaccid and dilated or abnormally-formed stomach. When the patient is in a *sitting posture,* the fluid reaches only the *lower segment of the flaccid sac,* and sprays the posterior gastric wall and the small curvature, also the region of the cardia and the fundus, slightly or not at all. If the patients with gastric dilatation who have become accustomed to the sound are made to lie down after an apparently satisfactory washing carried out in a sitting posture, and

lavage is continued while in the recumbent posture, surprisingl
tities of mucus and remains of food are brought up with the w
who have become accustomed to lavage in the recumbent po
very well, so that, after the stomach is filled with water, this
moved to and fro in the stomach by movement of the trunk.
wall is thus thoroughly washed up to the small curvature, w
ticularly important since this area is the preferable seat of
gastric diseases.

In hour-glass stomachs, those with deep diverticulum-like
by cicatrices, also in such as have a markedly developed antru
and antrum cardiacum, we sometimes observe that, after an ap
ough gastric lavage, turbid gastric contents which have been
sac suddenly pour into the funnel. The symptom may frequen
in diagnosis.

In removing the stomach-tube when the washing is fini
note that, with a lowered funnel, a column of fluid remain
because the gastric wall contracts firmly around the lower en
and closes the opening. By the siphon action, folds of muco
may even be drawn into the opening of the tube, and, shoul
the flow of water will cease suddenly with a jerk which may
throughout the entire tube. If, at this moment, the stomac
be rapidly withdrawn, there is great danger of *injuring the*
brane, or even of *tearing off a portion of it that has bee*
the tube.

This danger may very readily be averted by holding the
moving it, pouring a little water into the funnel which is rai
slowly withdrawing the tube, when water will again flow into
and the mucous membrane be forced away from the opening
When the tube is so far withdrawn that the opening is abo
we compress it in order to prevent any fluid from entering th
while the tube is passing over the epiglottis.

Immediately after lavage the patients should have some
then rest for a little while; they should make no exertion w
least an hour afterward.

Individualization and Interruption of Gastric Lavage.—
of character and will power exert quite an influence upon
operations, which, like most stomach washings, necessitate s
on the part of the patient. Where this influence is a delete
physician, by his personality and skill, may be able to ove
a time.

Moreover, there are definite pathological conditions to be
the case, the influence of which upon the course of the op
be foreseen nor always prevented, and, under some circumsta
be so great that lavage must be discontinued. We refer

the danger of extraordinary irritability of the gastric mucous membrane and the danger of hemorrhage.

According to experience lavage is best borne by patients with gastric dilatation, therefore by those to whom this operation is most necessary. The mucous membranes of the stomach and esophagus are so benumbed by the intense irritation of the stagnating gastric contents and by vomiting that they do not react to the irritation of the tube and the cleansing fluid. Hence the danger of injury to the mucous membrane by the stomach-tube in gastric dilatation is very slight.

The condition is different in irritable stomachs which are not dilated, which are sometimes even decreased in size (contracted or concentrically hypertrophied), which react by a decided, sudden contraction, by retching and vomiting, or by cough, to the least irritation; for example, to the slightest movement of the introduced stomach-tube, to a touch of the latter against the stomach wall, to the inflow of the fluid, or to the temperature or composition of the same. Vomiting in such cases often has a peculiar expulsive character. The water suddenly gushes from the mouth with great force so that care is necessary in handling the stomach-tube.

In such irritable stomachs another grave danger is that, after a sudden attack of retching with severe vomiting, or after an attack of coughing, the gastric wall is forced against the stomach-tube, and consequently *the mucous membrane is rubbed against the opening of the tube and becomes excoriated*, or *a fold of the mucous membrane is sucked into the opening and torn off*, or, if the stomach-tube has not been adjusted carefully enough, it is forced out of the stomach.

These stormy reactions of extremely irritable stomachs frequently cease if the patient obeys the command to breathe deeply and regularly. In other cases, the stream of luke-warm water flowing from the funnel held as high as possible quiets the irritable gastric walls. If this is not the case, it is risky to continue the lavage. It is wise to observe all precautions; we, therefore, slowly withdraw the stomach-tube, and defer the operation until another morning. Before attempting it again, we should never neglect to call the attention of the patient to the mistakes of the last washing, to caution him to breathe deeply and regularly, and to avoid this or that bad habit. If catarrh of the pharynx is present, this also should receive the necessary treatment.

In another category of gastric affections, lavage is disagreeable, and even dangerous, because of the *great tendency of the gastric mucous membrane to hemorrhage.* In the accidents during lavage which have been described above, hemorrhages may also occur, but only in consequence of previous gross mechanical injuries. Here we are dealing with *hemorrhages which occur without such injuries.*

There are certain catarrhs of the stomach in which the mucous membrane, particularly at the height of its folds, is enormously hyperemic.

The dilated capillaries of the mucous membrane mostly sh extreme permeability that quite insignificant variations in sufficient to cause mucous membrane hemorrhages. Among ma cases of lavage, I have known about ten such hemorrhages to though they were slight they necessitated the interruption o tion and, because they recurred with every attempt, I was discontinue lavage entirely in these cases.

In hysterical patients vomiting of bloody masses and of occasionally occurs without any loss of substance of the m brane. Therefore, in such patients also, we may be forced the stomach-tube on account of the hemorrhagic staining o the same is true of gastric crises of spinal origin.

Hemorrhages from esophageal varices must also be referr are more profuse than the previously mentioned form, and c ble the hemorrhages originating from gastric ulcers. In a not of this kind which was at first believed to be ulcer of the which, however, this diagnosis was abandoned on account of of all other symptoms of ulcer, the hemorrhages ceased entir influence of lavage. Here, apparently, the introduction of t pressed the ectatic veins of the esophagus. Similar conditi in hemorrhage from the ureter, where catheterization or sou a potent curative factor.

If, during lavage, *congealed blood is found in the stoma* observed in ulcerating carcinoma, in old ulcers of the stoma ticularly, where there is a stenosis of the pylorus with a s of the pylorus caused reflexly, producing hypersecretion, there *for interrupting the operation.* On the contrary, in cases of thorough emptying and cleansing of the stomach is an exce ficial measure, and well calculated to check an old hemorrh vent a new one. The empty and clean stomach is again ab properly, and by contraction the bleeding surface becomes borders of the ulcer are approximated, and the gaping lumi sels closed. The fresh, and often not inconsiderable, losses caused by the tearing off of particles of mucous membrane most distinctly show the mighty styptic influence of the r traction of the stomach. In the cases that have been reporte rhage has ceased rapidly and no ulcer formation has taken pla for the reason that the margins of the wound, in consequenc contraction, have approximated at once, have closed and heal tive wounds of the stomach, by first intention.

If, therefore, during lavage, a *fresh hemorrhage* should appear and be indicated by the red discoloration of the wat cian must have the presence of mind not to withdraw the s he must very gently, even though with some force, allow to empty itself, and thus make it possible for it to contract completely. Naturally the patient in whom this has happened must be treated for a time after his hemorrhage as in the case of ulcer.

The remedies used in lavage vary with the requirements of the individual case.

For simple cleansing of the stomach, lukewarm water is sufficient (at about the temperature of 86° F.); if, however, we wish to act upon the diseased gastric wall, additions must be made to the fluid, above all, soda for gastric catarrh, and table salt for deficient hydrochloric acid. A mixture of these salts is most serviceable:

℞ Sodium carbonate.................... part 1
Sodium chlorid..................... " 2
M. D. S.: To be dissolved in water (2 to 5 per 1000).

But even water alone may act in different ways upon the gastric wall; lower temperatures (less than 86° F.) stimulate and irritate; higher than 86° F., lessen irritation, have a soothing effect and relax. Under the influence of warm washings the pylorus opens, even when spastically closed, more quickly than under the influence of cool washings. If in the individual case, lavage of the stomach requires a long time, it is better for the patient not to have the water too cool, as thus too much heat may be withdrawn from him. Debilitated persons for this reason bear warm washings better than cool ones.

The pressure exerted during lavage has no inconsiderable influence upon the diseased gastric wall. In all the diseases in which lavage must be carried out cautiously, therefore in gastric ulcer and in corrosion of the stomach after poisoning with a tendency to hemorrhage and extreme irritability, the funnel must not be held too high,—not above the height of the patient's head. When we wish to produce a stimulating effect by the gastric washing (gastric douche), it should be held as high as possible.

After cleansing the stomach, *bitter tonics* are sometimes sprayed upon the mucous membrane, and are allowed to remain in the stomach in contact with the gastric mucous membrane for a few minutes; among these *quassia amara* (30 grams in half a liter of cold water, macerated overnight, and filtered early in the morning), *hop tea* and infusion of dried hops, and condurango are most recommended. I employ one to two teaspoonfuls of the fluid extract of condurango to half a liter of lukewarm water.

Abnormal fermentative decomposition in the stomach is best combated by thorough and regular lavage. We possess no reliable remedy for the relief of abnormal decomposition, fermentation and other processes of decomposition, which, without danger to the organism, can aseptically change the digestive processes in the stomach and intestine. In an abnormal production of organic acids in the stomach I have most often employed washings with a 1–1000 salicylic acid solution.

lavage are necessary the patient soon loses strength, the intestin receives sufficient fluid and nourishment from the stomach, t ments of the economy of the body can no longer be met, eve we use supplementary rectal alimentation, and unless an opera formed in such cases the patients slowly succumb to exhaustion

FUNCTIONAL DISEASES OF THE STOMACH

By H. LEO, Bonn.

The stomach differs from other organs in that it frequently presents subjective and also objective symptoms of disease which are not due to any organic change, or, at least, not to any which are recognizable by our present methods of investigation. In this respect, only the heart, that is, the circulatory apparatus, and the intestines show similar conditions. These clinical pictures may be grouped under the term, *functional diseases of the stomach*.

One of these groups presents well-developed anomalies of the gastric contents which are not, however, due to disease of the stomach wall, but to the ingestion of tainted food, or to the invasion of microbes which in themselves cause the decomposition of the ingesta in the stomach. These morbid conditions have been designated *dyspepsia*.

The second group is distinguished by clinical pictures which present either subjective symptoms dependent only upon alterations of the stomach or, combined with this, disturbances of gastric activity which may be objectively recognized. The majority unquestionably depend upon abnormal functions of the nervous apparatus, and these affections are therefore designated *neuroses of the stomach*, in spite of the fact that a direct relation to diseases of the nervous system does not always exist.

The differential diagnosis of individual diseases belonging to this group from one another and from other organic diseases of the stomach is not always easy. To accomplish this an investigation of the entire organism is, as a rule, necessary, and of the various disturbances which uncommonly often disturb the gastric activity, or may be due to abnormalities of the latter. Naturally the chief stress is to be laid upon the special examination of the stomach, in an anatomical as well as functional respect. On account of the increased importance of this in the last few years, it appears advisable to discuss somewhat minutely the best method of examination.

TO TEST THE FUNCTION OF THE STOMACH

Of the three functions of the stomach, the motor, the secretory and the absorbent, *the motor*, i. e., the intimate admixture and mechanical preparation of the food and its timely removal through the pylorus into the intestine is unquestionably the most important. The disturbances of this function, manifested by an increased or, usually, by a decreas which appear in the most varied diseases, are then conspicu degree. To differentiate them, besides other complicated an methods, it is sufficient to prove by the Leube-Riegel test-me or by the Ewald-Boas test-breakfast (see below) that th digestion deviates from the normal. As a decrease in the (*insufficiency of the stomach*) primarily occurs in gastric dila forego a full explanation of the test for this, and instead re to the article by Riegel. It is sufficient to state here that s a test-meal, and, at the latest, two hours after a trial-breakfas normally propels its contents into the intestine. If, therefor by sounding or by lavage ingesta are still found, the motility is diminished. On the other hand, experience teaches us tha under normal circumstances, three hours after a test-meal after a test-breakfast, still contains remains of food. If, a is already empty, we have proof of an increase of its motilit

While a test of the absorbent function of the stomach present time, produced no results which are of value in th gastric disease, the study of the *secretory function* by an e the constituents of the gastric secretion or of the admixtur with the intaken ingesta, and also of the normal and abno of decomposition, is of great practical importance. Only s ination of the gastric contents was incorporated into the di tric diseases have certain well-characterized clinical picture tion, hyperacidity, achylia) become known. Their results determine the diagnosis, either because we find changes in t tents which are characteristic of certain diseases, or beca this kind are absent; that is, normal conditions exist, and justified in excluding some diseases which come into questio ential diagnosis.

In regard to the *indications for the examination of the* this procedure, that is, lavage of the stomach, aside from th tions to be at once named, is absolutely harmless and, as carried on without difficulty, but is occasionally unpleasant tive patients, and, naturally, not in place when other diag mit us to make a positive diagnosis. The artificial removal contents is now only performed with a soft rubber tube, b contraindicated if hemorrhages have occurred even althou have elapsed since the last one. Otherwise, a fresh hem readily be produced.

The stomach contents are withdrawn either when the sto therefore, as a rule, early in the morning, or during the tion; if we suspect hypersecretion (see below) it is done a named. In these cases, we find a considerable amount of the stomach normally should be empty. Only when ingesta reach the stomach, that is, when present there, both in the healthy and in the sick, do we find besides hypersecretion a secretion of the gastric juice. In the examination of the empty stomach the proof of gastritis may also be valuable on account of the mucus present in the opening of the tube, and which contains profuse amounts of leukocytes and epithelium. This is also the case with the proof of insufficiency of the stomach, in which the stomach that should normally be empty in the morning still contains ingesta. In all other cases, we examine the gastric contents during the time of digestion.

During this process two objects must be borne in mind: First, the determination of abnormal processes of decomposition in the gastric contents, and secondly, the determination of the properties of the gastric secretion.

To determine the first, that is, the presence of abnormal products of decomposition or causes of decomposition, it is often sufficient if we obtain the gastric contents for examination upon the first visit to the patient, immaterial when and what the patient has last eaten. But it is advisable to do this only when we desire qualitative proof of the composition of the gastric secretion (hydrochloric acid and pepsin).

This, however, is unwise when an exact estimation of the activity of the gastric parenchyma is to be made, particularly if we wish to determine the proportion of the constituents of the secretion, and, especially, of hydrochloric acid. The composition of the gastric contents, which represent a mixture of the gastric secretion and the food that has been consumed, is dependent upon the amount and nature of the ingested food and upon the time which has elapsed since the food was eaten. Therefore, if we desire to ascertain the deviations of this composition from the normal, these deviations not being dependent upon accidental variations in the intake of food, but upon anomalies of secretion, we must be sure, in the first place, that the composition and amount of the introduced ingesta are always alike, and that the stomach contents are always obtained after the same lapse of time following the ingestion of food.

For this purpose it is, above all, necessary that the stomach be empty before the foods which are to cause a secretion of gastric juice are eaten. The best time for the so-called test-meal is, therefore, early in the morning, or in the course of the morning, several hours after breakfast has been taken. When it is doubtful whether the stomach is empty it must be washed out before the test-meal is given. Of the many test-meals which have been advised to stimulate gastric secretion, I shall here mention only the two which are in most common use, which are the best, and quite sufficient for all practical purposes.

Test-meal according to Leube-Riegel.—Toward midday the patient partakes of a plate of beef-soup, 150 to 200 grams of beefsteak, 50 grams of mashed potato and a roll. Four hours later, as a rule, the stomach is empty, but it is occasionally advisable to empty the stomach somewhat sooner or later. This method has the advantage that the gas membrane is stimulated in a normal manner, because all nec are contained in the meal. It should, therefore, always be em the simpler *Ewald method* gives a negative result in regard to acid and pepsin. In such cases we often note that the more prolonged stimulation of Leube's test-meal gives a conspicuou result; its disadvantage lies in the fact that, because of the co the components of the meal, complete uniformity of the same i can only be obtained with difficulty, and gastric patients in pa frequently unable to consume it entirely. In office practice unpleasant, but sometimes necessary, to evacuate the stomach after the meal, at a late afternoon hour.

Test-meal according to Ewald and Boas.—Early in the at another time when the stomach is empty, the patient rec two rolls of wheat bread (35 to 70 grams) and one or two without sugar or milk. Water may be taken instead of tea hour or an hour and a quarter (according to whether the sing quantity has been taken) the stomach is evacuated. This t the advantage of great simplicity, and the masses which are not have the smeary composition which they have by Leube's the evacuation may occur comparatively soon after the intake administration of the trial breakfast and the evacuation of contents may be undertaken during the office hour.

[The shredded wheat biscuit makes an excellent test-meal from yeast and contains the entire food elements of wheat. thoroughly masticated, and a glass or two of water form an meal.—ED.]

EXAMINATION OF THE GASTRIC CONTENTS

A. MACROSCOPIC AND MICROSCOPIC EXAMINATI

The *odor* of the gastric contents normally and also in most pa provided the stomach is empty before the test-meal is administered, tive. In stagnation of the ingesta (gastrectasis, dyspepsia, gastriti an expression of decomposition, the odor of fatty acids, acetic acid o (like rancid butter).

Blood is occasionally admixed with the stomach contents as a sm streak; it originates from retching, and is therefore of no pathogn tance. If, however, the entire contents have a distinct hemorrhagic the presence of an ulcer, carcinoma, or stasis in the portal vein sys and all further sounding is to be avoided.

Bile, characterized by its yellow or greenish color, is very frequentl gastric contents and is of no diagnostic significance.

Mucus in small amounts may also be found in the normal gastric quantity of hydrochloric acid present is slight. Larger amounts, par coagula, denote gastritis, unless the mucus originates from the respir In the case of gastritis, the microscopic examination of the mucus as stomach contents evacuated from the empty stomach is of importance. The microscope reveals numerous well retained leukocytes and epithelia; in acid gastritis in which the protoplasm of the leukocyte is digested, numerous cell nuclei are found while these normally are present in only slight numbers (A. Schmidt).

The *macroscopic composition of the ingesta* is of special significance. If, after Leube's test-meal, the particles of meat are split up, the production of hydrochloric acid is sufficient, perhaps even increased. On the contrary, if they appear unchanged there is a deficiency of hydrochloric acid, perhaps of acidity. Insufficient maceration of the fragments of bread favors hyperacidity as a greater amount of HCl inhibits the saccharifying action of the diastatic salivary ferment which normally occurs in the stomach.

The *microscopic examination of the gastric contents* may be of diagnostic weight on account of the previously mentioned formed elements in gastritis. The presence of large numbers of microörganisms denotes stagnation of the ingesta, but it must be remembered that the yeast cells may be due to the ingested bread.

B. CHEMICAL EXAMINATION

The gastric contents to be examined must be undiluted; except in an examination for ferments, it is unnecessary to filter them.

The examination primarily is for the acids, particularly hydrochloric acid, volatile fatty acids, and lactic acids. In most cases this is sufficient. Under some circumstances (if achylia be suspected), an examination of the ferment, perhaps also of the products of digestion, is expedient.

ACIDS

(a) *Qualitative Tests*

Reaction.—The evacuated contents of the empty stomach may normally have a neutral or even an alkaline reaction (the presence of bile and intestinal juices in the stomach). If, after a test-meal, blue litmus paper is not reddened, the condition is one of insufficient or absent hydrochloric acid secretion.

Hydrochloric Acid.—The test is first made with the *Günzburg reagent* (two grams of phloroglucin, one gram of vanillin, thirty grams of alcohol); a few drops of this with an equal quantity of gastric contents are placed in a porcelain dish and heated over a small flame. If a beautiful red margin appears, the presence of hydrochloric acid is proven. If, however, the red color does not appear, the absence of hydrochloric acid is by no means certain, as its presence, even in decided amounts, may be obscured by other combinations.

This error cannot occur in *the test with* $CaCo_3$; therefore, this test must always be used if the reaction with the Günzburg test is negative. For this purpose, we mix in a watch-glass some of the gastric contents with a pinch of powdered $CaCo_3$. The reaction of the mixture is tested with blue litmus paper, and this is compared with the original reaction of the gastric contents. If the redness of the litmus paper after treatment with $CaCo_3$ is less intense than before, or if no reaction occurs, the stomach contents (in the presence of volatile fatty acids and of lactic acid) contain hydrochloric acid. If the reddening is the same, before and after, no hydrochloric acid is present.

For further confirmation of this test *the digestion test* may be employed (see below).

Volatile Fatty Acids.—Several c.c. of the gastric contents are heated in a test-tube to boiling, while a piece of blue litmus paper is held over the test-tube. If the paper redden, the presence of volatile fatty acids is proven: if no redness appear, they are absent.

Lactic Acid.—About 10 c.c. of gastric contents are shaken up with about the same quantity of ether, and the ether which has taken up the greater part of the lactic acid present is poured off. To the ethereal fluid a few c.c. of a very dilute ferric chlorid solution are added (one drop of an officinal ferric chlorid solution to a test-tube full of water). Even if only minute quantities of lactic acid are present, upon shaking a distinct yellow color appears in the ferric chlorid solution.

(b) *Quantitative Estimation*

We must always remember that, in spite of many methods for this estimation, we do not determine the absolute amount of acids excreted in a certain time by the quantitative estimation of the acids, but we measure relatively the secretory function of the stomach, for the reason that we never know how much of the gastric contents has entered the intestine during the time in which we are evacuating the contents, and this, therefore, cannot be calculated. As, normally, there is considerable variation in the secretion of hydrochloric acid in the same and in different persons, we should only regard marked and constant deviations as pathologic.

If the presence of HCl and the absence of volatile fatty acids and lactic acids have been proven in the manner described above (which is the case in the majority of tests), this is sufficient to determine the total acidity for practical purposes. On the other hand, if we desire to learn the exact proportion of acids present, their estimation must be included. Upon the whole, although the qualitative proof of organic acids in fermentative processes is so important, their quantitative estimation is of no great practical utility.

Total Acidity.—To a definite quantity (5 to 10 c.c., although we may do with less) of gastric contents, to which a few drops of an alcoholic solution of phenolphthalein solution are added, a decinormal solution of caustic soda is added drop by drop from a graduated pipette until a red color appears and remains. The figure which is expressed by the amount of decinormal soda solution used in 100 c.c. represents the degree of total acidity of the gastric contents.

The normal values of total acidity usually vary between 20 and 60. If constan values below 20 are found, there is subacidity. If upon repeated examination value above 70 are noted, we are dealing with hyperacidity.

Hydrochloric Acid.—Of the many methods advised for the quantitative estimation of HCl I shall describe only the one proposed by myself which, among othe advantages, possesses that of great simplicity, and has shown itself, both in m hands and in those of others, to be absolutely reliable.

To 10 c.c. of gastric contents, a few c.c. of a concentrated $CaCl_2$ solution ar added, and after the addition of a few drops of an alcoholic phenolphthalein solution the mixture is triturated (as above) with a decinormal soda solution until permanent red color appears. Then, about 15 c.c. of gastric contents are mixe in a dry vessel with about 1 gram of dry powdered $CaCo_3$, and, after stirrin are filtered through a dry filter. 10 c.c. of the filtrate are measured off, and th CO_2 which has formed is evaporated by a current of air, when $CaCl_2$ solution an some phenolphthalein are again added, and the acidity is calculated by means of decinormal soda solution.

If the sum of the decinormal soda solution obtained in this tritration (calculat upon the basis of 100 c.c. of gastric contents) be subtracted from the number c.c. required in the first tritration, the difference corresponds to the amount normal soda solution required for the neutralization of free HCl present in 100 c.

f gastric contents. If the reaction for volatile fatty acids and lactic acids (see bove) is negative (as is the case in the overwhelming majority of instances), the gure obtained corresponds to the amount of HCl. This figure multiplied by 0.00365 ives the percentage in grams of HCl in the gastric contents.

If organic acids be present, 10 c.c. of gastric contents are again measured and haken in a beaker with about 50 c.c. of ether; the ether is then poured off, and this shaking and pouring off of ether is repeated five times. If the acidity of the residue of the stomach contents be then determined the difference shows the amount of the organic acids. This is subtracted from the total acidity previously obtained, and the remainder equals HCl.

FERMENTS

The presence of digestive ferments (pepsin and lab) may greatly assist us in the diagnosis of achylia (see below).

The test for *pepsin* consists in placing 10 c.c. of filtered gastric contents in each of two beakers, and also a flake of washed blood fibrin; to one of these a few drops of diluted HCl is added, and both beakers are placed in an incubator at blood temperature (98.6° F.). If, after several hours, the flakes are still present, in spite of repeated shaking of the beaker, pepsin and pepsinogen are absent from the gastric contents.

To determine the presence of the *lab ferment* about 10 c.c. of raw milk are mixed with 2 to 5 drops of gastric contents. Lab coagulation either occurs at once, or (at the temperature of the body) after a longer period. If the lab ferment is eventually absent, its prior stage, lab zymogen, may be recognized by adding 2 c.c. of a $CaCl_2$ solution to the previously mentioned mixture.

DYSPEPSIA

We employ the designation, dyspepsia, which in common parlance means "deficient digestion," to only a limited extent in that we restrict it to gastric digestion. But, even here, it is still further limited in that we exclude from this category organic diseases of the gastric walls which give rise to digestive disturbances.

In the term "dyspepsia" we include only those alterations of the stomach which are not due to an organic affection. It is true the ordinary conception of dyspepsia is decidedly more extensive, for we speak of dyspeptic difficulties when referring to abnormal sensations in the stomach without an anomaly of gastric activity being conjoined to them. This is especially true of so-called "nervous dyspepsia" (see below), the especial characteristic of which is that gastric digestion is quite normal. The contradiction in this is quite obvious, and although the designation which has become firmly rooted in our language is very difficult to displace, I nevertheless believe it to be correct in a systematic description to speak only of such conditions as dyspepsia in which gastric digestion is, in fact, disturbed.

By this classification, cases of dyspepsia are greatly limited. Since the gastric mucous membrane is too sensitive to retain for any length of time contents which have been subjected to decomposition without reacting in a short time by irritative processes, i. e., by catarr conditions belong in this category.

Under dyspepsia, therefore, and in a restricted sense, we acute dyspepsia, i. e., the digestive disturbances which the bu meets with daily, which are due to the ingestion of taintec generators of fermentation. As pure dyspepsia, without org tion, these disturbances will be considered only in their initia is, before a consecutive catarrh has as yet been produced.

To determine that only acute dyspepsia, and not gastriti an investigation of the gastric contents is always necessary. of products of decomposition, especially organic acids, volatil and lactic acid is not sufficient for this purpose. For eve gastritis, usually in consequence of anomalies of secretion abnormal change in the ingesta which become subject to acid True dyspepsia, however, is favored by the absence of dec of mucus, which, in acute gastritis, is always present in the tents, especially in the later stages; a microscopic examina the recognition of numerous leukocytes and epithelial cells.

The effect also of a single, thorough gastric lavage, whic and most certain *therapeutic remedy* in acute dyspepsia, aids nosis; for if, after this little operation, and without other re ures, restoration to health is immediate, we may feel assu gastric mucous membrane was not implicated, and that the p tion consisted in a decomposition of the gastric contents.

NEUROSES OF THE STOMACH

Neuroses of the stomach, with comparatively rare exception idiopathic. As a rule, they are caused by a general anoma tution, or, reflexly, are dependent upon disease of another orga cases, strictly speaking, we are therefore dealing only with of another disease which affects the stomach; the disturbanc activity are, however, frequently so prominent and characteris dominate the clinical picture, and for this reason alone they prehensive description.

The most important etiologic factor is neurasthenia, next well as chlorosis and other anemic conditions, diseases of th paratus (especially in females), wandering kidney and affe intestines, and these by no means exhaust the series of dis course of which functional disturbances of the stomach appear. they occur secondarily in cerebral and spinal cord affections, i tuberculosis, in peritonitis, in severe renal disease, etc. Nev making a diagnosis, we must consider their occurrence in v diseases.

SENSORY NEUROSES

Anomalies of the Sensation of Hunger.—These consist in a decrease (*anorexia*), an increase (*bulimia*), and perversions of the normal appetite (*parorexia*).

Anorexia, which is a distinguishing symptom in almost all gastric diseases, may also be autochthonous, and here, above all, psychical influences such as care, sorrow, pain, disgust, etc., which dispel the sensation of hunger for a longer or shorter time, play a part. Naturally, before making the diagnosis, the existence of an organic gastric affection must be excluded.

Parorexia, in which there is a tendency to eat improper things, such as sharp condiments, vinegar, iron filings, chalk, etc., while natural foods are declined, occurs particularly in women who suffer from chlorosis or who are pregnant, but these abnormalities may also occur in other hysterical and neurasthenic persons. The treatment is that of the fundamental condition.

In *bulimia* we are dealing with a pathological augmentation of the normal sensation of hunger. This varies extraordinarily under different physiologic conditions, and, particularly in growing persons, is frequently intensified; and, since food not only serves to maintain the healthy condition of the body but also to promote growth, we can only regard excessive increase of the appetite as pathologic. This is shown by an abnormal hunger which appears soon after profuse amounts of food have been taken, and, besides the unconquerable desire to eat, it is accompanied by numerous distressing symptoms. Among these are lassitude, numbness of the head, sometimes actual headache, specks before the eyes, tinnitus aurium, general sweating, and tremor as well as general weakness. In some patients local abnormal sensations, as in the region of the stomach, are prominent; these are sometimes designated as pressure, sometimes as actual pain.

As the sensation of hunger is produced by direct action upon the hunger center (central disease and chemical irritation of the center by an abnormal condition of the blood) as well as by reflex causes which may originate from the periphery, it is evident that the causes of bulimia may be extremely numerous. Besides neurasthenia the following must be mentioned: Organic diseases of the brain and psychoses, Graves's disease, diarrhea, helminthiasis, menorrhagia, pregnancy, diabetes mellitus, etc. Those cases of bulimia are especially interesting which depend upon other functional diseases of the stomach, particularly upon hyperacidity (see below) and, above all, upon hypermotility (see below); in the former case it is apparently the abnormal irritation of the hyperacid gastric juice, in the latter the abnormally rapid emptying of the stomach after the ingestion of food, whereby a stimulation of the hunger center is caused centripetally.

The *therapy* of bulimia, on account of the great difference in tl etiologic factors, must naturally be adapted to the underlying affectio The measures in the individual case are indicated in what has been pr viously said; in fact, in the case of helminthiasis, diarrhea, diabetes me litus, and menorrhagia, a rational treatment of the underlying affectio will in most instances suppress the bulimia, or, at least, decrease it. I hypermotility of the stomach it is wise to administer small quantities o food very frequently, and to alternate with fluid and solid food. In al cases in which there is hyperirritation of the nervous system, and thes form the majority, besides the general régime suited to this, the adminis tration of preparations of bromin as well as belladonna and similar reme dies is of value.

Gastralgia (*cardialgia, gastrodynia*).—This affection which, under th picture of gastric crises, forms such a prominent symptom in tabes dorsali and other central diseases, may also appear in consequence of the abov mentioned affections as a substantive disease. Yet, upon the basis of m experiences, the cases in which typical and marked attacks of pain in tl gastric region are observed, may only in an insignificant percentage l considered as gastric neuroses; even then the diagnosis is extremely que tionable, for the positive exclusion of cholelithiasis and gastric ulcer (som times also pancreatic calculi) is very difficult, and when we consider t great frequency of the two first mentioned affections, we had better, a doubtful case, incline to these, and adjust the treatment in conformit

Gastralgias of moderate degree, which cannot be referred to an orgar affection, are, on the other hand, quite frequent. In these cases the pa or sensitiveness is referred to the entire epigastrium, and this circumstan as well as the patient's frequent statement that the pains are lessened pressure, is of value in the differential diagnosis between ulcer and cho lithiasis; in regard to ulcer, the independence of the quality of the fo and of the time elapsed since the last ingestion of food also aids in t diagnosis.

Gastralgia is not infrequently combined with *hyperesthesia* of the ga tric walls, which is particularly observed in anemic and nervous individua and which shows itself by a more or less decided sensitiveness direct associated with the intake of food. This sensitiveness may reach a ve high degree, and cause severe irritative symptoms, such as convulsions a cataleptic spasms.

The main point in the treatment is to quiet the nervous system. I sides other measures, the constant current may be useful.

MOTOR NEUROSES

The disturbances of motility of the stomach consist in an increase diminution of this function, affecting the entire stomach, or only the car or the pylorus. There are, however, pathologic pictures which are di cult to explain otherwise than as a combination of irritative and depressing influences.

Atony (insufficiency) of the stomach, which appears so frequently in organic disease of the stomach (dilatation and gastrectasis), is one of the most important functional disturbances; it often occurs in anemia and other nutritive disturbances without demonstrable anatomic change in the gastric walls. In this condition we are dealing with atony of the musculature as well as a decrease in its elasticity and consequent impairment of the property of propelling the ingesta from the stomach at the proper time. As the abnormal tension of the gastric walls which results is usually only a transitory extension of the organ, I shall omit its consideration at this point, and refer the reader to the excellent article by Riegel.

Insufficiency of the Cardia.—While flaccidity of the musculature of the pylorus, causing an insufficiency of the pylorus, has only been described in isolated cases as a neurogenous affection (Ebstein), *insufficiency of the cardia* is an anomaly which may frequently show itself under different clinical pictures.

Where there is a moderate degree of atony and a simultaneous tendency to voluminous collections of gas which are sometimes increased by decided swallowing of air (Quincke), we also observe *eructation* (ructus, eructatio) of gases, which may vary in intensity, and be transitory or habitual; in the latter case this becomes very distressing to the patient. As the same symptom is conspicuous in almost all organic gastric affections, errors in diagnosis can only be avoided with care. Besides hysteria, a circumstance in favor of the purely nervous character of the affection is that the gas which is discharged is odorless and tasteless, and the eructation may voluntarily be suppressed by the impulse of the will. Besides general tonics, the *treatment* must be designed to influence the psychical element, and we must endeavor to strengthen the will power. Occasionally gastric lavage is of use.

Another typical clinical picture, which is attributable to incontinence of the mouth of the stomach, due not only to atony of the cardia but also to a flaccidity in opening and to antiperistalsis of the esophagus, consists in *rumination* (ruminatio, merycism). In persons affected by this condition, the ingested food rises from the stomach a short time after it has been swallowed, and returns to the mouth (*regurgitation*), where it is again chewed and swallowed. This process usually occasions no disgust, and since regurgitation, as a rule, occurs soon after eating (rarely later than an hour), the food has no sour, or even unpleasant, taste. Many patients are unconscious of any disagreeable sensation, while others are keenly alive to their repulsive condition. Not rarely they are able to suppress the regurgitation which they have voluntarily produced, but only temporarily, for this afterward recurs to an increased extent. I know several patients who have had this affection for decades, and who can prevent regurgitation by lying down immediately after their mea after some time, they rise, the regurgitation at once appears. cumstance that psychical alterations, fatigue, menstruation, etc. increased and more frequent appearance of the affection is i its nervous character.

The *treatment,* in the main, must be directed toward inc energy of the will and suppressing the pathologic process. Gas should be tried as well as preparations of bromin. Unfortunatel succeed in bringing about a cure.

So-called *nervous vomiting* may also be included in this cla tions. Vomiting is caused by increased muscular activity, but a contraction of the diaphragm and of the abdominal muscles immediately a compression of the stomach, that is, an expul contents, and it is unquestionably true that the forcing of t contents upward occurs all the more readily the less resistan on the part of the cardia. Nervous vomiting is characteristic the particular ease with which it appears (prodromes, especia are frequently absent), and it is obvious that flaccidity of the c an important rôle. This affection is prone to occur in hysteri also in chlorotics, more rarely in neurasthenics. The idiopat nervous vomiting described by v. Leyden, which is character periodic appearance (periodical vomiting), must be here inclu ous vomiting may exist for years without the nutritive condi patient suffering.

[Regurgitation of food due to stenosis of the esophagus, e the cardia, may be mistaken for vomiting. This is especially tr spasm. General nervous conditions may be the cause of the c and at the same time a nervous dyspepsia or a gastric ulcer A close attention to the character of the symptoms, especially of so-called vomiting and an examination of the expelled food the case plain.—Ed.]

In the diagnosis, an unusually large number of general af diseases of the most varied organs, intoxications and autointo well as organic gastric affections in which vomiting is a sympt excluded, and furthermore, particular stress must be laid upon factor of hysteria. Besides combating hysteria, local treatn stomach by lavage or irrigation and electricity are in place.

Hypermotility.—Hypermotility is a condition in which th tivity of the stomach, by means of which its contents are pr the intestine, is increased. In consequence the stomach is en a time in which, normally, ingesta should still be present. sufficiency of the pylorus also plays a causative rôle cannot be Hypermotility is by no means rare. It occurs particularly thenics, and shows itself by the appearance of bulimia (see the diagnosis, the proof that the stomach one hour after the Ewald, or three hours after a Leube, test-meal is empty or contains but few particles of food is necessary. In regard to treatment, see that indicated for bulimia.

Peristaltic Unrest (*tormina ventriculi*).—This anomaly is distinguished by increased peristaltic movement of the stomach (and frequently also of the intestine), and is accompanied by more or less loud rumbling which, with a thin abdominal wall, is distinctly audible. In making a diagnosis stenosis of the pylorus, above all, must be excluded. The *treatment* consists in the use of sedatives; occasionally lavage of the stomach acts favorably.

Spasm of the Cardia.—In contrast to the descriptions of this occurrence which are variously given, we must fully understand that the isolated spastic closure of the cardia may, primarily, cause difficulty in deglutition. We therefore have the symptoms of a deeply situated stenosis of the esophagus. This anomaly is purely functional, particularly in hysteria, but it also occurs in marked anemia without other nervous irritative phenomena. It may run its course with or without pain, it may occur paroxysmally, particularly in connection with the act of deglutition, or be permanent. Cases have been described in which the spasm had existed for years, and had given rise to a sac-like dilatation of the esophagus above the stenosis (Leichtenstern, Dinkler). Whether, however, in these cases an ulcer of the cardia did not originally exist would be very difficult to decide. For the diagnosis, examination with the gastric sound is decisive. Upon its introduction, if a spasm at the height of the cardia exists, resistance is met with which, as a rule, is readily overcome. This procedure may be accompanied by pain or occur without it. In the absence of pain the diagnosis is easy. In a differentio-diagnostic respect, carcinoma, in the usually youthful patient, need scarcely be considered. But with a painful course of the affection, care must be taken not to confound the condition with ulcer of the cardia. The successful introduction of the sound would exclude the latter, while, on the other hand, treatment similar to that for ulcer is to be instituted.

Besides general remedial measures, sounding the esophagus is indicated. Sometimes a single sounding is sufficient. If this is not the case it is well to apply cocain by moistening the lower end of the tube with a solution; or, still better, after introducing the gastric tube to the depth of the cardia, a few cubic centimeters of a not too concentrated cocain solution may be poured into the tube, which is allowed to remain *in situ* for a few minutes.

Spasm of the Pylorus.—This frequently occurs in organic diseases of the stomach, and is said to be also purely functional, particularly in hyperacidity. The consequences are stagnation of the ingesta and tympany of the stomach, and the latter may be particularly severe *when combined with spasm of the cardia. Asthma dyspepticum,* so designated by Henoch, is due to a combination of this kind.

cant, on the other hand, is the fact that toward the end of digesti microscopic examination of the remnants in the opening of the t numbers of cell nuclei are found, since, normally, but few nucl kind are present. Another circumstance that favors an existin is that upon withdrawal of the tube from an empty stomach adhe ticles of mucus containing large numbers of leukocytes are found.

The views in regard to the best *treatment* of hyperacidity ha gone decided change. The fact that an increased amount of H gastric contents unquestionably disturbs the saccharification of containing 'starch by the salivary ferment which is swallowed appear rational to limit starchy food in the diet of the patients from hyperacidity (to the utmost), and to place them upon ani Experience has, however, shown that a diet of this kind does n lessen the difficulties, but not infrequently they increase, and this with the fact that objective investigations of the gastric content decrease in the amount of acid. In the first place, a meat diet is not well borne by patients with gastric disease, and, above all, a in albumin causes a greater secretion of acid (Hemmeter, Fle gensen and others). It is, therefore, rational to prescribe for t a mixed diet which possesses the property of combining acids, creases acid secretion to the slightest degree, and is as little ir possible. These indications are best fulfilled by milk, and by m fat, since fats have an extremely slight influence upon the gastric milk is declined, or not well borne, as is unfortunately often the these patients, lime water may be added to it. Soft, pappy rice stewed calves' brains, sweetbreads, etc., are given. Toasted wh and tea with a plentiful addition of cream, are likewise perm also prescribe soda bicarbonate, magnesia (hydrated), Carlsba other alkalies, besides narcotics (especially belladonna). In there is a favorable influence from takadiastase, whose saccharif erty is less disturbed by hydrochloric acid than that of ptyalin. tion, stimulating measures are not to be neglected.

Hyperacidity is a very stubborn disorder, and we must, th guarded in the prognosis.

Hypersecretion (*flow of gastric juice*).—This affection is ch by an abnormally profuse secretion of the gastric juice, which o out the stimulation of the ingesta. It may be continuous or pe may run an acute or chronic course. The amount of HCl of tion may be increased (gastrorrhœa acida) or normal. In case the symptoms are those of hyperacidity; but, even wh not increased, the patients complain of pyrosis and acid Vomiting of a more or less profuse, watery, acid fluid while t is empty of food is characteristic of the condition.

The diagnosis depends upon the last mentioned symptom the results obtained by the stomach-tube while the stomach is empty; that is, whether by this procedure large amounts (100 c.c. to 1 liter) of an almost clear fluid having an acid reaction and containing HCl and pepsin are evacuated.

The treatment must be directed to the removal of the probable underlying affection, neurasthenia, anemia, etc.; the preparations of bromin, narcotics (codein, belladonna, cocain, etc.), are in place as well as irrigation of the gastric mucous membrane.

NERVOUS DYSPEPSIA (GASTROSIS)

Among the gastro-neuroses, so-called "nervous dyspepsia" occupies a peculiar position. Leube, who accurately portrayed the clinical picture after it had been described by others, particularly by English authors (Beard and Rockwell), defines it as a pathologic condition in which subjective symptoms of chronic dyspepsia, analogous to those of chronic gastritis are present, but in which objectively demonstrable anomalies of activity, as well as organic changes of the stomach, are absent.

It is evident that, in a pathological condition the basis of which is subjective difficulties, the negative results of investigations of the anatomical and functional disturbances of the stomach create a difference of opinion among authors in regard to the definition and the limitation of the affection, and that many deny that there is any justification for considering a symptom-complex of this kind as a disease, *sui generis*. In fact the number of authors who are opposed to the pathologic conception of "nervous dyspepsia," in Leube's sense, is quite considerable. We would digress too far, and in entire opposition to the character of this article, were we minutely to consider the comprehensive literature of this subject. Some of these explanations bear a remote date, and it may be assumed that not a few of the opponents of "nervous dyspepsia" have, in the course of time, made subsequent observations, and have changed their former opinions.

According to my experience which coincides with that of others, there is no doubt of the existence of a clinical picture as described by Leube and others. Far be it from me to maintain that, in such cases, an organic change can always be excluded. We are unable, however, to demonstrate it, and we may often convince ourselves that no organic disease known to us is the cause of the affection. Shall we therefore deny the existence of this clinical picture? With as much right as we could eliminate many other affections, such as migraine, neuroses of the heart and of the intestines, flittering scotoma, pruritis, etc., from pathology. For in these conditions also we are unable at present to detect any anatomical anomaly which is certainly the cause of the symptoms or to exclude it. The justification and the necessity of limiting "nervous dyspepsia" as a disease, *sui* *generis,* as well as the previously mentioned functional disturb depend upon experience, in consequence of which the symptor and those still to be discussed appear, and must be consic expression of this affection of the stomach.

Objections have been raised to the description of the cli as well as to its name. This is quite reasonable. Since turbances of digestion are absent in the disease it would nat that the name "nervous dyspepsia" would provoke discussio also be added that the nomenclature does not correspond t conditions, since the dyspeptic difficulties are, without exc attributed to an implication of the nervous apparatus. In ı in a majority of the cases, this origin may be at once admi for those in which the disease depends upon neurasthenia organic disease of the nervous system, or where it appear neurosis, particularly from the sexual apparatus. Even whe tive factor is looked for in intoxications or infections, such of nicotin, alcoholism (here it is true there is usually chror syphilis, malaria, etc., which usually exert a damaging in the peripheral nerves, we are justified in ascribing to the p the development of the gastric symptoms. On the other h be remembered that the symptom-complex peculiar to thi appears in consequence of anemic and general debilitative co as chlorosis, the puerperium, improper and insufficient f would seem far-fetched to look for the cause of the morb in the nervous system. This would be more obvious in a nutrition of the parenchyma of the stomach which is undoub which, however, as the actual causal factor, has as yet sh pathologico-anatomical changes of the organ. For this reas nations neurasthenia dyspeptica or gastrica (Burkart, Ewal be regarded as improvements; for, as v. Leyden quite prop the chief disturbances are not in the stomach, as should be the other names which have been proposed, the best appears bined gastroneurosis," which, it is true, is somewhat com does not emphasize the sometimes non-nervous nature of the

Perhaps it would be wise to choose a more indifferen which would, however, permit us to recognize that we are a functional anomaly of the stomach. As such I propo *Gastrosis.* The designation nervous dyspepsia has become so in our minds that it will not readily be eradicated, but th subordinate significance provided we are unanimous as signifies.

Among the etiologic factors which have been mentioned which is important because of its comparative frequency. abnormal state of nutrition due to the fact that the period individual meals are too long. There are some persons who, like dogs, take only once a day a meal which really deserves the name, and yet they continue in the best of health. In spite of the latter circumstance, there can be no doubt that this habit is irrational. It is contrary to the laws regulating the function of the stomach as a reservoir of food, and it cannot be an advantage to the stomach to remain empty for several hours during the day and then suddenly to become excessively loaded. The effect of this is particularly noticeable in persons who do not voluntarily or from habit make these long pauses between meals, but are forced to it by the stress of circumstances. This occurs in very busy physicians, particularly surgeons, in merchants, in lawyers, etc. According to my experience, this category of occupations furnishes the main contingent of the cases of gastrosis. The circumstance that the digestive disturbances of the patients improve when they change their mode of life and take more frequent but smaller meals, is proof of the etiologic importance of this improper mode of nutrition.

The *symptoms* of nervous dyspepsia, as already stated, are in the main subjective. The patients complain chiefly of a feeling of pressure and distention in the gastric region, and of eructations which sometimes become exceedingly severe and loud. The appetite, as a rule, is disturbed. The patients have more or less repugnance to food, and not rarely complain of nausea. Tendency to vomit and actual vomiting also occur, but not so frequently as in gastritis. The loss of appetite in some cases depends upon autosuggestion. The patients fear that by eating their distress will be increased, and therefore refrain from food as far as possible. On the other hand, even bulimia occurs; the patients then are tormented by a voracious appetite which causes them to crave food. As a rule, as soon as they begin to eat, the bulimia not only disappears, but also all desire for food. They can only take small amounts, and are unable to satisfy the existing hunger. Perverse anomalies of taste are also frequently observed. Acids and other foods difficult of digestion are especially preferred, and in the circumstance that, after their use, the preceding discomfort often lessens, this clinical picture differs from other organic affections.

Among the abnormal sensations which originate from the gastric region, besides fulness and eructation, pyrosis must be mentioned. This symptom, which is an almost invariable accompaniment of actual dyspepsia, of "sour stomach," is not always found hand in hand with an increase in the acidity of the gastric contents, nor need this be the case in nervous dyspepsia. There are, however, instances in which the degree of acidity exceeds the normal limits.

Actual pain in the stomach is but rarely complained of, and, even then, it is not the pain upon pressure which is so characteristic of ulcer. The gastric region may be sensitive to the pressure of palpation, but often this is only an indefinite, disagreeable feeling, which is also observ sensitive patients whose stomach and other abdominal organs a normal.

The condition of the bowels varies. Constipation exists in as in other chronic gastric affections. Diarrhea is rare, an anomaly is present.

The disturbances of the general condition may be manifol spond to the varied etiologic bases of the affection. From th of neurasthenia (such as numbness in the head, lassitude, hypochondriasis, even melancholia, on the other hand frequ irritability, cardiac palpitation, vertigo, etc.) it is not always cide whether we are dealing with coördinated symptoms of the of the disease or with the sequels of the gastric affection.

The general nutrition may also vary according to the c In most cases the patients are well nourished, and there is sign of suffering. But we also see patients who are hollow-ey or less emaciated with no malady save one of the stomach for the disturbance. The history then reveals that the emac to insufficient food. These patients, partly from fear of di in obedience to unwise professional advice, have for a long only a minimum of food, and have actually undergone a sta It cannot be wondered at that, in consequence of this limi the resulting loss of resistance of the walls of the stomach, t instead of decreasing have been decidedly aggravated. These hopeful cases for treatment; the physician who clearly r affection and acts upon this knowledge, helped by the previo with ease, bring about what seems a miraculous cure.

In the *diagnosis* of nervous dyspepsia, which should be ma care, we must first decide whether the complaints of the patien attributable to gastric activity, or whether the difficulties r stomach are not due to a disease of other parts of the organis they do not at least owe to it their origin. In this connecti be too skeptical, since such so-called dyspeptic symptoms as sized above may occur in a number of different affections be minutely described at this point.

The presence of well developed neurasthenia or hysteria, that these anomalies existed prior to the dyspeptic sym importance in the diagnosis. It is obvious that these will the diagnosis, as neurasthenic and hysterical persons may al organic disease of the stomach, and as gastrosis also occurs i in the main, show no functional disturbances of the nervous

The coincidence of dyspeptic disturbances belonging to complex of nervous dyspepsia, with functional disturbance without organic disease, is also important in diagnosis. C tion and irregularity of the cardiac action in connection with gastric affections are, therefore, symptoms which must be considered in the diagnosis.

The report of the patient that the digestive symptoms vary, that, instead of the usual disturbances, there are times when the disease seems to have almost disappeared, particularly when the mind is diverted by pleasant company, may be of value. With Rosenheim I admit this fact, but it rarely occurs, and is not absolute proof that other gastric patients, for example, those suffering from chronic gastritis, occasionally will forget their dyspeptic disturbances when in interesting society, at the theatre, etc. Nevertheless this occurs, although less frequently.

The objective examination of the stomach is chiefly important because conspicuous anomalies are not found. I am of the opinion that the moderate grades of gastroptosis which prevail so commonly in women without giving rise to symptoms, cannot annul the diagnosis: Nervous dyspepsia.

In regard to the functional test, Leube, as remarked above, has laid special stress upon the fact that the motor function is intact. According to my experience, we must in general agree with him, and the cases in which there is a well developed slowing of motion should not be considered as nervous dyspepsia. Moderate grades of atony, or the presence of small remains of food two hours after a test breakfast and seven hours after a test-meal, belong to the same clinical picture, particularly if this condition is not constant. In testing the functions of the stomach these frequently great variations are characteristic, and this is true of a higher degree of secretion. In this respect I entirely agree with Boas and Rosenheim. It is true that while the degree of acid is usually within normal limits we occasionally note surprising variations of the acidity below and above the normal.

On account of this variability of the symptoms, and the difficulty of limiting exactly the clinical picture, we should never fail to consider the possibility of organic disease of the stomach before we make a positive diagnosis.

The condition may be mistaken for *gastric ulcer,* for this affection does not always present the well known classical symptom of typical pain occurring a short time after the intake of food, but it may develop without any pain, and an examination of the gastric contents may reveal no characteristic differences. If there is actually well developed pain, the case is not one of nervous dyspepsia. Marked tenderness and a drawing sensation in the gastric region, however, are observed, and the report that these sensations are not increased by external pressure but are rather relieved is decidedly against ulcer. The manner in which the sensitiveness of the gastric region shows itself upon palpation is important. If it be localized to a circumscribed area this favors ulcer, while a diffuse, painful sensitiveness of the entire epigastric region points to the nervous character of the affection. If hysteria is present we not rarely observe, especially

in thin patients, painful pressure areas upon both sides of tl
column. But a confusion of this previously mentioned pain up
with simultaneous gastroptosis is not impossible.

Even if there is no marked sensitiveness of the gastric regi
not at once exclude ulcer, particularly when dealing with youi
As the examination of the gastric contents permits no posil
sions, it is wise in cases of this kind to proceed as if ulcer w
therefore, to prescribe absolute rest, fluid diet, and silver niti
this means the affection is not favorably influenced, the ass
gastrosis is justified.

Gastric carcinoma may be excluded with certainty provid
peptic difficulties have existed for a long time without any di
the body-weight. We must, however, remember that carcinon
a very slow course without producing symptoms other than tl
pepsia, anemia, and slight emaciation. In elderly individua
be very cautious in the diagnosis of an affection of this charac
for a short time, perhaps only for a few months. If well deve
toms of neurasthenia or hysteria have preceded, they favor t
of nervous dyspepsia, although, of course, patients of this k
attacked by carcinoma. A normal hydrochloric acid reaction
sence of lactic acid in the stomach contents are factors whi
favor nervous dyspepsia. Absolute certainty, however, can onl
when regular weighings of the body, under sufficient nourishm
loss in weight.

Well developed *gastrectasis* presents such a characteristic
ture that this disease can scarcely be mistaken for another. I
readily occur when the symptoms depend upon a simple *aton*
tric walls. Here definite conclusions are reached by the exa
the motor function. The proof that there is no stagnation of
that, on the contrary, the stomach discharges its contents n
cludes this anomaly. Naturally, we should not be content v
examination, for, as has been stated, variations in the motor fu
in nervous dyspepsia. But the existence of this affection is r
if, transitorily, a prolonged retention of the ingesta in the
observed.

Chronic gastritis must be specially borne in mind in th
diagnosis, as the subjective symptoms of this condition comple
with those of nervous dyspepsia. The absence of a coated ton
non-appearance of mucus in the vomit and evacuated gast
particularly when the stomach is empty (see above), favor tl
gastritis is present, upon microscopic examination of the co
tube we find profuse formed elements, leukocytes and epith
chemical examination of the gastric contents after a test-meal
portant, for, as a rule, the secretion of HCl is deficient in ga

is, of course, not always the case, for in so-called acid gastritis, which is rare, it may even be increased. Therefore, in cases of this kind, the circumstance that the affection occurs in neurasthenics or hysterical persons, as well as the modification of the disease by treatment, is of significance.

The *prognosis* is always doubtful. By proper treatment many cases are favorably influenced and even complete recovery ensues. But it sometimes happens that, at the onset, decided improvement takes place, and the easily excited patients are enthusiastic regarding the result; then, however, relapses occur which affect the entire nervous system, and reproduce the former gastric disturbances. In other cases all endeavors favorably to influence the condition are without avail. These cases test the patience of the sufferer and the physician.

In *therapy* the psychical treatment is hardly less important than the somatic. It is true we will only rarely be successful if we represent to the patients that the affection is a very slight one, which may yield to a little exercise of the will. As a rule the patients resent a suggestion of this kind. It is in crass contrast to their discomfort, and, as a matter of fact, the mere determination not to be ill rarely suffices to cure the disease.

But there are instances, as stated before, in which a mere change in the diet, i. e., a return to the usual home living, is sufficient to restore the patient to health. These are, however, not cases due to neurasthenia or hysteria, but those which at the onset were very insignificant gastric difficulties, but excessive carefulness and a fear of the injurious effect of wholesome and plentiful food has limited the diet to such uniform and insufficient food that it has not conduced to improvement; hence, in consequence of imperfect nutrition of the walls of the stomach and of the entire organism, a steady aggravation of the dyspeptic symptoms has been brought about.

A., a manufacturer, aged 45, as a young man had disease of the left pulmonary apex, which, however, entirely disappeared and there is no trace of it at present. After this illness, the patient became extraordinarily careful and anxious about his health. The slightest deviation from the normal made him fear the onset of disease.

According to report, several years ago he began to have a sensation of fulness and pressure in the epigastrium after his principal meal, accompanied by eructations and constipation. Gastritis was diagnosticated, and the patient, whose appetite was always normal, limited to an extreme the quantity and quality of his food. By this means and the use of Carlsbad water, his condition is said to have at first improved, but gradually there was an aggravation, and, although pressure in the region of the stomach was no longer especially noticeable, the eructations continued, and to these vertigo, headache and palpitation of the heart were added. Appetite was lost, and not infrequently the monotonous diet caused repugnance. Nevertheless, the patient continued upon this diet through fear of injuring himself by a change.

The examination of the powerfully built, but pale and poorly nourished patient, revealed no anomaly of the stomach (total acidity upon repeated examination 30 to 60, Günzburg's test positive, motility normal) or any other part of the organism.

I explained to the patient that there was no disease of the stomach, nor, in fact, any organic affection, and that his symptoms were due to an injudicious mode of life, particularly to monotonous and insufficient food. I, therefore, advised him not only to take the "easily digested foods" but to return to his former favorite foods. No restrictions, whatever, in diet were necessary. The patient at first was somewhat incredulous but followed my advice and at once ate some peas with sour kraut and pickled meat. As the symptoms which he looked for did not appear he gained confidence, again ate and drank like a healthy man, and after three weeks presented himself to me completely cured. His weight steadily increased (about 21.9 pounds) and except for constipation, which still existed, all symptoms had disappeared.

Cases of this kind are, however, infrequent, and, particularly when dealing with nervous persons, treatment such as above described is not in order. Here, above all, it is necessary to tone up the entire nervous system. Bodily and mental rest, removal from the ordinary occupation, are usually the first requirements, but by no means always. Patients who have previously passed their time in idleness, with hypochondriac thoughts dwelling upon their stomach affection, should be advised to take up a stimulating occupation, strengthening and even fatiguing bodily exercises. But the diet must be regulated. In severe cases, particularly in debilitated women, complete isolation and rest in bed, at least for the greater part of the day, and forced feeding, will be beneficial.

In other cases a change of air, i. e., residence in pure fresh air (medium altitude in the woods) is sufficient. Special stress must be laid upon the stimulation of the skin by baths, massage and electricity. Above all, the diet must be regulated. Food difficult of digestion, and particularly that which causes flatulence, must be avoided, but the fact should be emphasized that the diet should be varied as much as possible, and the intervals between the meals are not to be too long.

Drug treatment, compared with these general dietetic rules, is of decidedly secondary importance, but it is frequently necessary to support the latter mode of treatment. Here stomachics such as rhubarb, tincture of quinin and bitters, iron and Fowler's solution, perhaps also quinin as a tonic, then sodium bromid and valerian, as well as various remedies for the usually stubborn constipation, are beneficial.

THE DIAGNOSTIC AND THERAPEUTIC SIGNIFICANCE OF SECRETORY DISTURBANCES OF THE STOMACH

By H. STRAUSS, Berlin

The significance in diagnosis and therapy of the secretory disturbances of the stomach is unquestionable, and is important from a scientific as well as a practical standpoint. This will be admitted even by those who hold the certainly justifiable view that the nosological significance of disturbances of secretion was greatly over-estimated in the first few years of the new departure in functional diagnosis of the stomach. But there is no reason for under-estimating it to-day. Though we must acknowledge from actual observation that a person absolutely without secretion in the stomach may not only live for years but even decades without showing noteworthy disturbance of nutrition, we know, nevertheless, that the result of marked secretory insufficiency of the stomach—as, for instance, diarrhea—may damage nutrition to a marked extent, and that, on the contrary, excessive secretion—continuous secretion of the gastric juice—by causing chronic under-nutrition, dryness of the tissues, and perhaps also withdrawal of chlorin may induce conditions which threaten life. It is not my object to describe in detail these conditions in which disorders of secretion play a rôle, but I shall consider secretory disturbances only in so far as they represent subjects for *diagnostic* and *therapeutic* consideration. If this manner of presenting the subject is *a priori* unfamiliar to the reader accustomed to regard definite clinical pictures as nosological entities, it certainly will facilitate the description, and the physician who understands also other disturbances of the stomach, their importance, and the methods for their recognition, will not be led by such a description to a too one-sided conception of the subject, or allow himself as a therapeutist to drift into errors of omission. As a rule, the careful physician will have already grasped the knowledge that, as a basis for the erection of pathologic pictures in gastric pathology, the clinical symptom-complexes which we meet to-day in gastric pathology frequently do not equal the pathologico-anatomical changes with which we have long been familiar. Following O. Rosenbach, almost all investigators in this realm have come to the conclusion that for the purpose of treatment the interests of our patient demand not only a thorough investigation of the symptoms of *anatomical* change (position, form, and size of the stomach, production of mucus and pus, superficial investigation of the products of secretion of the gastric mucous have been detailed to a great extent in the article by Professor L tional Diseases of the Stomach" (page 66). I shall not consum their description, but shall allow myself only a few critical rem upon personal experience. At the close of this article I shall e few methods not given in the previously mentioned treatise.

Test for Hydrochloric Acid.—For the test of *free* hydrochl see no reason to reject Congo paper, since this permits in the sin ner the sufficiently positive recognition of its presence. Th amidoazobenzol paper recently advised for this purpose by Ein not find superior to Congo paper when I conducted investiga eight years ago in Riegel's Clinic. Only when Congo paper giv results, should the Günzburg reagent be employed. For the estimation of free hydrochloric acid I still advise dipping with according to the processes of Riegel, Mörner and others—perh utilization of dimethylamidoazobenzol as an indicator. With reagent, which also contains phenolphthalein and dimethylami I have not always obtained a definite result. The question in free hydrochloric acid should be determined *quantitatively* can, ion, only be decided in practice in the individual case, since th who frequently investigates the gastric contents learns in th time, from the combination of values for total acidity, from reaction, as well as from other signs in the gastric contents, wh conclusions are to be drawn in the majority of cases. From m rience I maintain this in regard to the determination of *comb* chloric acid, and I have only rarely found a strict necessity fo tative estimation—at least for practical purposes. In the o majority of cases in which there is a deficiency of free hydro and the absence of lactic acid, the comparative estimation of tion of total acidity and the degree of digestion in the evacua enable us to form an opinion sufficiently clear for practical pu the amount of secretion and the measure of the peptic labor of provided we start with the assumption that when large quanti bined hydrochloric acid are present in the evacuated contents, a of digestion may, as a rule, be recognized, while this is absent are only slight quantities of combined hydrochloric acid. I can strongly recommend the complicated methods which are advis mining combined hydrochloric acid, as their adoption into pra with great difficulty, and the exactness of their results is oft mensurate with the complexity of their use. If we desire amount of secretion while excluding the phosphates, we may phenolphthalein and tincture of cochineal (see later), or Leo's be employed, or a trial may eventually be made with a met advised by Cohnheim and Krüger. In my experience, howev necessitates the utmost care, and gives only approximate, not always absolutely exact, values for combined hydrochloric acid if, as I found when working with Dr. Cahn, at the same time, investigations are made of the gastric juice for free hydrochloric acid, utilizing Leo's process. The previous method, advised by v. Noorden and Honigmann, for "hydrochloric acid deficit," is simple but does not always give similar values in regard to the amount of combined hydrochloric acid in a test-meal at the height of digestion, as I determined after numerous investigations of the gastric contents for free hydrochloric acid with Leo's process, and with Congo titration; Ehrmann determines it at from 0.05–0.07 HCl—particularly in cases of stagnation with good secretion—and also higher values are observed.

Total Acidity.—The total acidity of the gastric contents, as is well known, is determined by the aid of phenolphthalein which reacts upon all three of the acid components of the normal gastric contents (free and combined hydrochloric acid as well as acid phosphates). For the determination of the "physiologically active" hydrochloric acid alone this property of phenolphthalein is unsatisfactory, especially in the cases in which, by a diminution of the production of hydrochloric acid, the amount of acid phosphate in proportion to the total quantity of the "physiologically active" hydrochloric acid increases. The acid phosphates, as I am in a position to prove, form no inconsiderable part of the total acids. In 14 investigations (according to Leo's process) I found that the total quantity of acid phosphates in the empty stomach, that is, in the secretion free from food, amounted in value to 7.5 (maximum 10, minimum 5); in 19 test breakfasts with free hydrochloric acid, an average value of 11 (maximum 15, minimum 8); and in 14 test-meals with free hydrochloric acid, an average value of 22 (maximum 38, minimum 9). Therefore, another indicator would be desirable which, without reacting upon the phosphates in the absence of lactic acid, would show exactly what acid factors of the gastric contents might be referred to hydrochloric acid production. With this in view I have attempted the comparative titration of about 30 gastric contents with methylene orange and tincture of cochineal, and in the investigation of 7 cases of typical "apepsia gastrica" with phenolphthalein, I found upon an average an increase of about 16 in the value of the total acidity in comparison with the values obtained with cochineal tincture. In 15 cases in which stagnation was present the differences were even greater; they amounted to between 18 and 21. In about half the cases these differences were compensated for by the values which I obtained with Leo's process, in the other half this difference was increased to a slight extent, and therefore we may say that among the conditions found in the stomach the cochineal tincture does not reveal acid phosphates. In my opinion, the influence of acid phosphates also deserves mention because of the values for total acidity which we obtain after administering a test breakfast and test-meal at the height of digestion, and because, as I was

(gastrosuccorrhea). Expressions such as hypochylia and hyperch
are formed from achylia, as little convey the true meaning as do
achylia, since chyle is entirely different from the gastric juice.
heterochylia is quite misleading as it does not describe a substan
logic picture. On the other hand, it would be well to retain
like hypopepsia, apepsia and hyperpepsia for the reason that t
express the *effect* of gastric digestion without revealing more i
the nature of the secretory disturbance than terms which deno
of hydrochloric acid secretion. This is self-evident when we
the amount of hydrochloric acid contained in the gastric juice is
tant and *decisive* factor for gastric digestion, because even a g
rich in pepsin is incapable of producing a corresponding diges
without a large amount of hydrochloric acid. These designati
suitable for the reason—as I have already remarked—that spe
ances of pepsin, lab-ferment, steapsin and the diluting secret
yet not been so fully recognized as to evoke any special therape

SUBACIDITY (APEPSIA GASTRICA)

In occupying ourselves first with cases of **hypochlorhydria**
hydria, that is, **hypopepsia** and **apepsia,** we observe that they
comprehensive clinical picture, but, instead, a series of peculi
mon to the gastric contents. Among these the absence of free
acid is the most important.[1] Other peculiarities, such as the
mentary character of the evacuated gastric contents which,
apepsia, reveals only traces of oral cavity digestion—not gastri
showing low values for total acidity, usually a high specific g
values for the dextro-rotary substance, the absence of stain
of amylolysis as well as the appearance of shreds of structu
upon microscopic examination of the gastric contents, are as
of the decreased secretion merely of symptomatic importan
occasionally be of value in a clinico-diagnostic sense. Of pe
ferment production we may say in general that they are mo
than hydrochloric acid secretion, but, like the steapsin produ
plete insufficiency of secretion, these may also be decidedly

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[1] Naturally there are also cases with abnormally low total acidity
hydrochloric acid is still present, and also cases (rare) with norm
and absence of free hydrochloric acid without lactic acid. As in the
they are usually associated with disturbances of motility in patien
tion is decreased to only a slight degree—the secretory energy of
insufficient for the production of the *physiologic plus* of free hy
these cases are looked upon as instances of "relative" subacidity,
normal stomach at the height of digestion must show a physiolog
hydrochloric acid.

entirely absent. According to Glaessner, only in carcinoma of the pylorus with complete or almost complete absence of pepsin may the distinct presence of lab-ferment be observed. That we miss a constantly recurring parallelism between acid values and pepsin is, in my opinion, explained from the fact that, besides other causes—according to the experimental investigations of Pawlow, Schiff, Herzen and others, there are various stimuli which promote in a one-sided way hydrochloric acid or pepsin production—in examining the total acidity we have considered too little the influence of phosphates, whose amount depends only partially upon the energy of secretion of the affected stomach. To this may be added that in former researches with the Hammerschlag process, the inaccuracy of the method has not been sufficiently taken into account in judging the results. In apepsia gastrica, at a time in which individual authors found different values for pepsin; I employed the Hammerschlag process with my own modifications, and in 4 instances I twice found the peptonizing ferment positively absent, and twice a property of peptonization of from 10 to 12 per cent., while on using the Mett process in 8 cases of apepsia gastrica, I found the property of peptonization only twice and then respectively 2 and 4 mm. In regard to the "diluting secretion," I proved in earlier investigations of the specific weight, and in more prolonged studies of the osmotic pressure of the gastric contents, as well as in quantitative estimations of the excretion of water by the gastric walls, that even in extreme secretory insufficiency, and especially in apepsia gastrica, it was still present, although apparently in diminished amounts.

In regard to the degree of total acidity in apepsia gastrica, I must reiterate what I have previously stated of the importance of phosphates in an estimation of the total acidity of the gastric contents. Although, as a rule, the majority of my cases of apepsia gastrica showed values for total acidity between 3 and 7—in a few cases I found the gastric juice neutral with phenolphthalein—I have, nevertheless, repeatedly observed cases with an acidity of 10 and 12, which possessed no property of peptonization, and at most showed only traces of combined hydrochloric acid (Leo's process). I have, therefore, made it a rule in suspicious cases of apepsia always to titrate with litmus and with tincture of cochineal.

As apepsia gastrica represents the highest grade of subacidity, so this affection distinctly demonstrates the clinical expressions of subacidity, its effects upon gastric and intestinal digestion, and upon metabolism; hence, in this description, I shall enumerate the causative factors and also the therapeutic indications which arise when a secretory insufficiency of the stomach is determined.

SYMPTOMS

The clinical signs which we observe in the higher grades of subacidity vary greatly. Often symptoms are entirely absent. At other times we meet with indefinite complaints of sensations of pressure or of eructation, partly of loss of appetite, partly of intestinal di particularly of diarrhea—or of more or less painful sensat abdomen. The great variety of clinical pictures in which w acidity as a symptom is not surprising when we consider tha in a mild or severe degree accompanies or follows in the tra maladies. Thus we find it not only in numerous local gastr among which carcinoma and alcoholic gastritis are probably tl quent, but also not rarely in nervous and organic general aff ticularly in cardiac and renal disease, in pulmonary tubercul the various disturbances of metabolism. For example, in the tory of 30 cases of persistent and "uncomplicated" apepsia ga I observed for a long time, and of which I possess accurate were 8 cases of chronic alcoholism, 4 of pernicious anemia, 5 pulmonary tuberculosis, 3 of chronic nephritis, 1 of Graves' well-developed chronic hysteria, 1 of trauma, 2 of severe er gastric mucous membrane (in both cases there were simultan toms of stenosis of the esophagus leading to a "stenosed" form In 5 cases no especial etiology could be discovered. I have transitory forms of apepsia gastrica in neurasthenics, above a of cases of delirium tremens. When I refer to an "uncompl of apepsia gastrica, in contrast to a "complicated" form, I larly in mind that prognostically ominous form which is an ac of malignant neoplasm.

DIAGNOSIS

As it is especially important in the absence of a recogn and metastasis (if necessary, palpation per rectum and X-ray of the mediastinum)[1] to base the differential diagnosis on *th* *the gastric contents,* I shall briefly include in this descriptior diagnostic criteria, above all, the significance of an *admix* *with the stomach contents,* and the possibility of evacuating b when the stomach is empty. In more than 6 cases which, observation, were positively recognized as non-carcinomatou trica, I found blood in the stomach empty of food, and in detected shreds of tissue; therefore, I maintain that *only th* *of blood and pus indicates carcinoma.* As to the diagnostic i the presence of shreds of tissue, I agree with Kuttner. Ir in which I repeatedly found shreds of tissue upon lavage stomach, the patients complained of a gnawing sensation, pain, and, upon taking food, of even more severe symptoms sidered hemorrhagic erosions which, in common with Ewald and Boas, and in opposition to Einhorn, Parish, and others, I found in various gastric conditions, but I am not inclined to believe in the uniformity of a special clinical condition in hemorrhagic erosions. An increased vulnerability of the gastric mucous membrane I have quite often observed in cases of apepsia gastrica as a consequence of chronic nephritis, or as an accompaniment of chronic alcoholic intoxication. As apepsia gastrica may also be observed as a secondary accompaniment of true pernicious anemia, the differentiation of the forms of apepsia gastrica occurring in connection with carcinoma may, under some circumstances, be of practical significance. Here it must be borne in mind that pernicious anemia going hand in hand with apepsia gastrica, in contrast with carcinoma ventriculi, will, in spite of the severe anemia, usually present a well maintained panniculus adiposus, and that a thorough examination of the blood will generally reveal decided differences between these affections. For those cases of pernicious anemia in which there is no profusion of megaloblasts in the blood, I especially advise the minute observation of the leukocytes; for, in my numerous studies of pernicious anemia, I have found that with a diminution in the total number of leukocytes, which is quite usual, there is a relative increase of the mononuclear cells, while in the majority of cases of carcinoma, leukopenia is more rare, and the multinuclear cells are perceptible in normal or even increased amounts. Anemia without severe cachexia—it is true this is scarcely ever observed with the typical blood picture of pernicious anemia—may also occur in carcinoma of the lesser curvature which leaves both ostia of the stomach intact. In cases of this kind the nutrition of the patient is often but little disturbed, although hemorrhage from the ulcerating tumor may produce the picture of anemia (often to only a slight extent the picture of cachexia). In my experience cases of this kind are by no means so rare as we might suppose from the reports of various authors; for, among the cases of carcinoma which for several months I had opportunities of examining in the Clinic,—in the majority of cases, up to the time of death or of the operation,—more than one-third ran their course without symptoms of stenosis, and especially without the finding of lactic acid.

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[1] Glandular tumors of the mediastinum have been actinographi by Weinberger (see his Atlas) and others.

Practical interest—particularly therapeutic—is also elicited by those chronic intermittent diarrheas which, in my experience, are noted in about one-third of all cases of apepsia gastrica. We have long been familiar with these diarrheas, as well as with their relation to severe forms of subacidity, the condition having been lately described by Einhorn, Oppler, and others. In some instances they represent the most striking and unpleasant expression of the secretory disturbance, and they chiefly require observation for the reason that they may decidedly damage the nutrition. In my opinion, the tendency to diarrhea depends to a great extent upon the fact that the coarse ingesta, on which the stomach exerts no digestive effect whatever, mechanically irritate the intestine, and also up that a larger number of bacteria with noxious influence upon t have reached this organ than is the case with a normal secretic the virulence of one or the other of these bacteria has been Moreover, the diarrhea may cause an injury to the general nut a disturbance of motility is associated with apepsia gastrica, the ingestion of food insufficient because of vomiting, etc. T a disturbance of motility combined with apepsia gastrica, tha high degree of subacidity, becomes apparent when we examine contents from the fact that lactic acid may appear in such gr as to be easily recognized by the method I have indicated, wi is well known, we can only determine pathological amounts o in the test breakfast or in the residue of the stomach before f That the absence of pepsin, as was first maintained by Hamr necessary for the development of lactic acid, besides the cor subacidity and motor insufficiency, I regard as not only unp present time, but as most unlikely; for, under my direction, v. that in the artificial conditions of experiment, and under c otherwise favorable for the production of lactic acid, pepsin inhibits the appearance of lactic acid fermentation. Only latel an opportunity of observing three cases of apepsia gastrica wi but undoubted lactic acid. Two of these cases occurred in following cholelithiasis, and were confirmed by operation. T occurred in an elderly colleague who, since he was first e years ago, has continued in the best of health. I formerly observations in a case of complete erosion of the stomach wit tion of numerous cicatrices and "stenosing pyloric hypert case of duodenal stenosis in consequence of tubercular perito as in a case of fat necrosis of the pancreas. Notwithstandi quite ready to admit that the finding of lactic acid in non-m ditions is so rare that, if continuously found for weeks, I co ploratory laparotomy to be not only justified but sometimes e necessary, provided we cannot certainly exclude carcinoma. own observations of the frequent occurrence of lactic acid differ from the ordinary in that I succeeded in demonstratin in only about 60 per cent. of my cases of gastric carcinoma particularly emphasize that among these cases were a large nu an autopsy was conducted.

In regard to the cases in which an absolute cessation o acid secretion did not occur, the presence of combined hyd even without a quantitative estimation as I have previously be assumed in all cases in which the total acidity, without la present, reaches a certain amount; i. e., with the employm phthalein it exceeds 20, and, at the same time, the gastric positive signs that gastric digestion is present. If, after a test breakfast, the gastric contents without free hydrochloric acid show an acidity which exceeds 30, and if the examination for lactic acid, which should never be omitted in any case of subacidity, gives a negative result, we are probably right in assuming the presence of combined hydrochloric acid. As I have repeatedly observed in cases of stagnation combined with slight subacidity, in rare cases without the presence of lactic acid, higher values of total acidity—values from 50 to 60—may be found. I noted this particularly in cases in which ulcer of the pylorus gradually underwent carcinomatous degeneration, but also in non-carcinomatous cases of pyloric stenosis with a secretion which was not quite sufficient. In these cases, the gastric contents did not show the symptomatologic and simultaneous increase of the long bacilli which indicates lactic acid. Cases of this kind have not only confirmed my view that combined hydrochloric acid may inhibit lactic acid fermentation, but by their excellent chymification, as well as by their amount of erythrodextrin, they have proven that where large amounts of combined hydrochloric acid are present, notwithstanding the absence of free hydrochloric acid, we have the objective signs of proteolysis and of disturbed amylolysis. In regard to chymification, I must here remark that occasionally, even when large amounts of lactic acid were present, I succeeded in recognizing a slight degree of chymification in the gastric contents.

TREATMENT OF SUBACIDITY

Since it is my intention in describing the treatment of subacidity to consider the diminution of the secretory energy of the stomach only as a symptom of definite affections, I shall not fully discuss the treatment of these maladies, but shall limit myself in the main to the therapeutic indications necessary to combat this condition.

Diet.—Beginning with the dietetic treatment, we find, fortunately, entire unanimity between theory and practice. This is first shown by the requirement that the food be finely divided, and of such a character that it will pass the pylorus as under normal circumstances. The importance of this therapeutic principle, so long established in practice, we can appreciate after we understand that the reduction of bread and pastry to a fine, pappy mass is not only the function of mastication in the oral cavity, but also that of proteolysis in the stomach. As I have demonstrated by microscopic investigations, a small portion of bread (consisting of gluten albumin) may be compared to tissue or a sponge in whose meshes the starch granules are deposited. To secure this reduction of food with defective proteolysis, good teeth are absolutely necessary, therefore in many cases of extreme subacidity the examination of the teeth by a dentist and the correction of defects form the first part of the treatment. The thorough cleansing of the oral cavity is also necessary, for the reason that the stomach of the patient in question can offer but slight resistance to the bacteria

descending from the mouth. In patients suffering from subac
ough mastication is also necessary for another reason. Recen
tions (Pawlow, Schüle, Troller, Riegel and Schreuer, as well
periments conducted by Martin Cohn under my direction) have
in cases in which the property of secretion has not been entir
is capable of increase upon decided stimulation, thorough ch
in fact, increase the production of acid. Therefore, slow eatin
ough mastication are to be especially insisted upon with those p
suffer from subacidity. As a rule, crisp bread and pastry whi
ground up by the teeth, such as crackers, zwieback, toast, etc., a
The more tender varieties of meat are given, such as poultry, v
place of that containing coarse fibers held together by a tough
tissue network (beef, lamb), and we should see that the food is
in the kitchen as to facilitate its mastication. According to th
tions of A. Schmidt, in which I entirely concur, too thorough
meat, that is, broiling which hardens the connective tissue w
subsequently dissolved in the intestine in apepsia gastrica, may
intestinal wall and thus produce diarrhea. Raw meat should i
to the patient suffering from subacidity. Gelatinous foods (
very useful since gelatin dissolves into fluid in the stomach. In
in which it is impossible to administer sufficient nourishment in
described, as well as by the contingent use of artificial foods,
ment of means for disintegrating food, for example, Collin's a
masticators, or small meat choppers, are advisable, and it is
patient to use the apparatus himself since food brought upon
a broken-up condition is less stimulating to the appetite than
in its original form and then finely divided by the patient hi
point, in my opinion, is all the more worthy of consideration
preparation of food and its appetizing appearance are impor
to a patient suffering from subacidity. Pawlow has shown tha
ance of food has a stimulating or depressing effect upon the ap
materially influences the intensity of the secretion of gastric
may truly say that the sight of appetizing food not only make
water, but also causes the gastric juice to flow, and to the
gourmand, we maintain that food eaten with zest is half digest
reason, in all cases of subacidity in which the glandular parc
reacts to stimulation by a secretion of gastric juice, great we
attached to the attractive and appetizing preparation of the f
obvious from the fact that many of these patients already suf
of appetite. Therefore spices, meat sauces, and the other c
the kitchen—within certain limits—are just as necessary
stomachics of the apothecary. In regard to the latter, I mus
passing that they act upon the nerves of smell and taste and
a feeling of desire which reflexly influences gastric secretio

Drug Treatment.—Drug treatment of subacidity represents
hand a substitution therapy, and on the other hand an attempt
the diminished secretion. Substitution therapy aims to supply
acid and the ferments which have been lost—particularly p
without, and probably represents, so far as the introduction
concerned, the oldest form of organotherapy upon an exact b
If we study the consequences of subacidity from the introducti
chloric acid and of pepsin, a critical judge will not place a h
upon the effect of such treatment; for, as the investigations
Schüle and others have demonstrated, the amount of hydro
combined by the proteids compared with the quantity of hydr
therapeutically administered by the mouth is so great that th
hydrochloric acid can hardly be looked upon as a substitute f
tion which has been lost. Moreover, in regard to pepsin it m
that, with adequate quantities of hydrochloric acid, even sli
of pepsin are enough to produce a satisfactory proteolytic effect
ever, the introduction of pepsin is useless if, during the entire
digestion, a sufficient quantity of hydrochloric acid is simultane
in the stomach. Benefit from the various wines of pepsin, there
ably because of the stomachics they contain. In cases with c
of secretion it is far wiser to use the pancreas preparations wh
a neutral or alkaline floor and permit digestion of the small
begin in the stomach; that is, where gastric digestion is lacki
digestion is aided by the administration of proper preparati
these preparations *pankreon* has been most serviceable in my
from papain, which is said to be active in an acid media, I
remarkable results nor have Grote, Hirsch and others. In
special form of substitution therapy, the administration of c
juice, which was first advised by Pawlow and subsequently by
French investigators—in Germany this has only been emplo
Meyer—I have no personal experience. I do not believe, howe
method is often absolutely necessary, nor, the material being
obtain, that it will come into general use.

Great value was formerly attached to certain products t
secretion, and Pawlow by masterly investigation and technic h
it possible for us accurately to study this question experimenta
found that raw meat, meat juice, meat broth, Liebig's meat
gelatin, certain peptones and large quantities of water stimula
juice. Herzen compared dextrin (not chemically pure) and
extract, and found that the combination of large doses of the
grams) in a dog with gastric fistula stimulated the gastric
formed pepsin. Employed alone, dextrin particularly influ
formation, while Liebig's meat extract chiefly acted upon the
Alcohol proved a mighty stimulant to the gastric secretion b

lutely no effect upon the production of pepsin. Alcohol administered per rectum also showed itself a stimulant to the gastric juice, as Metzger determined in Riegel's Clinic. Herzen reports that he produced favorable results in man with a mixture of Liebig's meat extract and dextrin in a palatable form. Another preparation, inulin, after the administration of which Herzen found a marked amount of pepsin in the gastric juice if he simultaneously administered alcohol, I have employed in a few cases in the form of a tea containing inulin (25 grams in 300 c.c. of the tea for a trial breakfast). Although I have in a few cases seen a slightly stimulating effect upon the gastric juice, yet, considering the expense of inulin, this was not so marked as to tempt me to make very extensive experiments.

It may be urged in opposition to the explanations given here that there are cases of apepsia gastrica which run their course for years and even decades without any marked disturbance, and we may conclude from this that it is not always necessary in cases of subacidity to follow the outlined treatment here suggested. In reply it may be maintained that no proof has yet been furnished that the persistent functional over-exertion of those glands of the intestine which furnish the proteolytic ferments and their combinations will never produce injurious effects. What we have stated of the relation of certain forms of diarrhea to apepsia gastrica, and the favorable influence upon them of measures here described, by no means strengthens the arguments of those who declare these measures to be unnecessary; and even if we could adduce no positive facts in support of the fundamental principles here developed, the purely theoretical assumption that prophylaxis is the best therapy is sufficient reason and justification. This is particularly true of the suggestion of small and frequent meals, since these, more than any other means, conserve the motility of the stomach, the weakening of which at once disturbs the equilibrium of the patient's metabolism. A disturbance of motility in cases of apepsia gastrica soon changes the stomach from a natural disinfectant apparatus into an incubation apparatus for various microörganisms—under some circumstances, also the pathogenic—and, from a teleologic point of view, a reason may be discerned in the facts that the stomach in apepsia gastrica usually empties itself more rapidly than under normal circumstances, that it is unable to digest its contents, and also that it has no power upon micro-parasites, but rapidly propels them into the small intestine, in which a great number of microörganisms appear to perish.

HYPERACIDITY

In the description of an *immoderate* secretion of acid we must sharply differentiate those conditions in which gastric juice abnormally rich in hydrochloric acid is excreted *only by the stimulation of the ingesta* from those in which gastric juice is excreted with a *permanent secretion—one independent of the introduction of food.* We must, therefore, sharply distinguish hyperacidity (superacidity, peracidity) from hypersecr persecretion, parasecretion, gastrosuccorrhea). As in the former ach reacts only to the stimulation of the ingesta present, and] secretion powerful in digestion, in the second case a secretion t without stimulation, and, as to cause and effect, the stomach in case bears the same relation to the latter as a pump or open artesian well. But since it is not my purpose here to discuss tion in how far hyperacidity is a substantive disease or is merely of other affections, I shall at once define our chemical conceptioı acidity. To express this in figures is very difficult, for, on the the amount of secretion varies greatly under physiological coı one and the same individual as well as in different persons, a other hand, the sensibility of individual persons to an excess chloric acid in the stomach shows extraordinary variations.] this latter factor merits special consideration because only tl suffering from hyperacidity seeks a physician who suffers dis account of his disturbance in secretion. In my opinion, ther scription of hyperacidity necessitates the simultaneous delinea those factors which produce a more or less marked acid hyperesl sides, our conception of the condition, that is, whether we bas ical conception of hyperacidity on the total acidity, or make chloric acid most prominent, varies greatly. Among those wh the first theory, Ewald estimates hyperacidity in a total acid 60 and 70, and Rosenheim above 60; Johnson and Behm consid juice with a total acidity of 70 as hyperacid, and Boas regards light a gastric juice with an acidity of over 2 per thousand. Ri laying much stress upon the total acidity, also attaches great to the proportion of free hydrochloric acid. "The most imp is the free hydrochloric acid. Only where this is increased are in speaking of an actual hyperaciditas hydrochlorica. Value 80 and more after a test-meal, of 50 to 60 and upwards after a t are frequently found here." Schüle considers the gastric juice acid when it reaches a height of 0.22 per cent. more than free acid, and shows a total acidity of more than 70. Schneider m a gastric juice with more than 0.25 per cent. of free hydroch to be looked upon as hyperacid, and that we may also regard a gastric juice which still gives a reaction for free hydrochloı a dilution of more than ten times its bulk. Although it must without more ado that gastric contents which with a total a give an intense reaction for HCl may be called hyperacid, yet cases I should be reluctant to diagnosticate hyperacidity of the upon this single symptom of the gastric contents, but only u of peculiarities which the test breakfast or the test-meal enabl nize. For we may examine gastric juice in which titration

as to the influence of motility in hyperacidity. For a long t
remarked that one hour after administering the test-breakfast
hyperacidity, and upon the evacuation of the gastric contents, a
amount was not rarely evacuated. This fact was long known t
only recently relinquished the antiquated method of estimatin
amount of gastric contents solely by expression, that is, by aspir
out subsequent lavage. For more than eight years, after ever
tion of the gastric contents I have made an exact estimation of
of the gastric contents by the aid of a mathematical formula wh
on the relation of the specific gravity of the undiluted gastric j
of the gastric juice diluted with 100 c.c. of water, this pri
learned subsequently, having been previously employed by Jaw
scientific investigations. I still employ this mode of calculat
the estimation of acidity according to the method of Mathieu a
in cases of anacidity leaves us in the lurch; that is, only by the
of a complicated modification given by Cohnheim are we enab
a conclusion. It may be interesting to note here that the amou
contents I have obtained in a great number of investigation
persons one hour after a test breakfast reached on an averag
of 150 c.c., while in 40 cases of hyperacidity I obtained an av
of 210 c.c. (the lowest being 124 c.c., and the highest 400
trary to numerous authors, I do not believe this increase in
of gastric contents one hour after the administration of the test
at least in uncomplicated cases—to be the consequence of
"atony" (an expression which had best be eliminated from th
ture of gastric pathology since it is not only superfluous but, in
of the dissimilar significance attached to it by different auth
to cause confusion) but to be the product of an increase in the
the gastric juice of a hyperacid stomach. In numerous resea
complicated cases of hyperacidity I was unable to detect any s
insufficiency either by the currant test, by the fermentation test,
scopic examination of the evacuated gastric contents. Perh
which has already been alluded to, that the "layer test" of
usually gives a lower layer quotient than in subacidity, and t
ally a conspicuously low value for the molecular concentration
contents is found ("hydrorrhœa gastrica") may be utilized
sense as my explanation. The view that the increase of the ga
in hyperacidity is to a great extent the consequence of an in
tion is somewhat favored by the experience that, in contrast
tion, just described, of the test breakfast (rich in starch), u
the test-meal relatively rich in albumin and poor in carb
extremely rapid emptying of the stomach may frequently be
phenomenon may be satisfactorily explained by the theory
breakfast, by the too early interruption of amylolysis ("Se

lta"), and the disturbed liquefaction of the introduced starches, promotes n increase of secretory stimulation. Johannes Müller admits that the igestion of bread is generally delayed by a decidedly acid secretion, and hat the inhibition of amylolysis appears to be influenced more by the rapidity than by the intensity of the acid secretion.

SYMPTOMS

As already indicated, the clinical manifestations of hyperacidity vary in individual cases to an extraordinary degree. One case will run its course without symptoms, and another with excessive pain which occasionally reaches such a degree and is so frequent that signs of chronic under-nutrition and loss of strength may appear. Complications such as erosions, fissures with consecutive pylorospasm, ulcer of the stomach, and especially ulcer of the pylorus, may in isolated instances impress their stamp upon the case. However, except for such "complications," in the great majority of cases of hyperacidity we may construct a clinical picture which, although not applicable to all cases, nevertheless applies in a great many. The patients are usually thin—but not always—are, as a rule, between 20 and 40 years of age, the majority of them belong to the erethismic type, they complain of gnawing, painful sensations of pressure in the gastric region, and state that these unpleasant symptoms occur as a rule several hours after eating—after the midday meal, or in the late afternoon hours—they continue for a time and then disappear. The mechanical state of the food is said to be unimportant, and the pains to disappear frequently by the ingestion of milk or by a large dose of sodium bicarbonate. The appetite is good, but the patients refrain from eating through fear of pain (cibophobia). Vomiting is rare, but eructations are frequent, as well as nausea and retching. The patients usually complain of chronic constipation. Provided complications or other diseases which, according to experience, play a rôle in the etiology of hyperacidity (pyloric stenosis, gastroptosis, hernia epigastrica, etc.) are not present, objective investigation in most cases reveals only slight diffuse sensitiveness on pressure. Frequently even this is absent.

Hyperacidity is observed in different countries in varying frequency. According to Jaworski it occurs in Lemberg in 51.8 per cent., according to Einhorn in New York in 51 per cent., according to Johnson and Behm in Stockholm in 36.4 per cent., according to Kövesi in Budapest in 30.4 per cent., according to Mathieu and Rémond in Paris in 29 per cent., according to Bouveret in Lyons in 25 per cent. of all cases of gastric disease which come under observation. In my own experience it is noted in Berlin in scarcely more than one-third of all gastric cases. At all events, among the clinical cases received at the Charité in Berlin, the disease is decidedly less frequent than it was in the Clinic in Giessen, as I had an opportunity of observing.

ETIOLOGY

The causes of hyperacidity also vary in the individual cas
partly of a local, partly of a general, nature. Among the lo
chemic, thermal and mechanical irritants play a rôle which, upo
action, may perhaps also lead to parenchymatous changes in
mucous membrane. I believe it very likely that the constant
rich in spices and the generous consumption of alcohol—especi
Rhine wine—play a rôle in the etiology of hyperacidity, and
cognizance of the thermic factors because hyperacidity is rela
mon among bakers, who, as is well known, frequently eat
Moreover it is by no means certain that the frequent drinking
fluids upon an empty stomach does not predispose to hypera
addition there is a large group of local and general neuroses
toxic (tobacco!), or arising reflexly[1]—and, what I believe to
of serious discussion, the chronic constipation so often noted in h
With their removal the symptoms of hyperacidity frequently
By some authors (Hemmeter) continuous over-stimulation with
is considered of special etiologic importance, and Westphalen e
inates a congenital predisposition attributable to the predom
diet of preceding generations. Cloetta has also shown that in
same litter, those nourished with meat show free hydrochlori
a meal while in dogs fed with milk it is absent. Although C
find no absolute difference in the histologic structure of the m
brane of these dogs on different foods, I believe it at least
organic changes may be produced in the gastric mucous m
hyperacidity. There is, as I have proven in several of my c
acid gastritis, i. e., a combination on the part of the stomach
duction of mucus with that of hyperacidity. Such cases upo
the empty stomach not only show more mucus than in the norm
water of a normal stomach before food is taken is grey, has a tu
ance, and resembles the water after cleansing the mouth—bu
scopic examination of the peculiar mucus plugs from the test-b
shows a conspicuous number of glistening nuclei of leukocyte
pletely digested protoplasm. The conclusion is obvious that
of acid production in such cases is not only functional but,
anatomical, especially in the cases of hyperacidity running
with motor insufficiency, and in which the autopsy reveals pro
the glandular parenchyma.

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[1] Some cases of hyperacidity and gastroptosis, hernia epigastrica,
tonitis may also be of more or less neurogenic origin.

TREATMENT OF HYPERACIDITY

In the treatment of hyperacidity diet occupies the first place. Its importance is evident from the fact that, probably, in no branch of the treatment of gastric affections are the fundamental laws of nutrition so warmly disputed as in this instance. The question chiefly resolves itself into this, whether preference should be given to albumin which readily combines acids, or to starches which are less stimulating to the production of hydrochloric acid in the patient suffering from hyperacidity. To-day, when the waves of discord have become somewhat smoother, we may say that occasionally in this discussion the point of view has been too one-sided, inasmuch as it has been overlooked that in the dietetic treatment of the patient the entire organism is to be considered as well as the stomach, and that in the consideration of dietetic experiments and experiences, meat and albumin have often been regarded as synonymous with carbohydrates and vegetables. In some of the earlier researches, too, the importance of the employment of "equicaloric" amounts of ingested food, and differences in the composition of the food, have not been sufficiently taken into account in the individual experiment, as is apparent from the special preparation of the food, its volume, and the labor necessary in masticating it. Finally, in my opinion, the varying etiology of hyperacidity from the basis of nutrition has been too little considered in the individual cases. A certain bias of observation has until recently been manifest, for, in the question under discussion the cry has almost always been "here albumin" or "here carbohydrates" while of fat we have only been told that patients suffering from hyperacidity "may" have good butter, or that large quantities of fat should be avoided for the reason that they readily disturb intestinal digestion by the hyperacidity of the gastric juice. Fat, at the present time, not only occupies a position of equal importance with the two other nutritive products in the diet of the person suffering from hyperacidity but even a preferred position, for the reason—as we know from the pioneer experiments of Pawlow upon the dog, and those which I simultaneously carried out with Akimow Perez, and the subsequent ones of Backman and others in man—that fat inhibits gastric juice secretion, and therefore is utilized normally in the intestinal canal of the patient with hyperacidity, yet does not inhibit the motility any more than an equal quantity of other caloric food products. The *curative* power of large quantities of *milk fat* in the dietary of hyperacidity thus appears to be proven, accordingly fat, especially milk fat, has been accorded a permanent place in the treatment of hyperacidity. In earlier investigations which I made regarding the relation of the dextrorotary substance in the human gastric contents, I found in concentrated solutions of sugar another dietetic measure to diminish gastric juice secretion. Similar researches have recently been made by v. Aldor in man, and by Clemm in the dog, with gastric fistula, and the results have been confirmed. For the removal of the acid symptoms, concentrated tions are most effective if administered to the patient only appearance. Instead of sugar solutions we may use honey, a Clemm has shown, levulose lacks the powerful, inhibiting secr of dextrose. For the rational treatment of hyperacidity, howe measures to diminish gastric juice secretion are by no means su we must simultaneously remove from the diet all those articl themselves possess the property of increasing the secretion of juice. As food and dainties in this respect have already bee in the description of subacidity, I desire only to urge the utn the free use of extracts of meat (concentrated bouillon, sauce spicy preparation of food in general, above all, such food produc prolonged mastication. A rational *arrangement of the meals* to be highly important in hyperacidity. It is directly opposed conception of the nature of hyperacidity that a patient with t should take frequent but small meals. The essential feature of consists in an abnormal irritability of the secretory apparatus ach, the expression of which is not manifest during the peric stomach rests, but only during its time of labor. For this rea tain, with A. Schmidt, that but a few and—provided there ar factors to the contrary—somewhat larger meals are indicated.

In regard to the *mixture* of individual food products, I those who have lately recommended a vegetable or a lacto-vege in the treatment of all those cases of hyperacidity in which to be the consequence of a functional neurosis, or in which th of the latter at least dominate the clinical picture. Accor experience, in the remaining cases, the administration of a is best,—one which, in a normal calory amount, contains con (from 150 to 180 grams)—especially milk fat, such as cr Jaworski's "kraftmilch," or Gaertner's fat milk, fat cheese, oi to Walko, even by the stomach-tube), emulsions of almond not bacon and beef fat—as well as a normal quantity of album ing to about 120 grams) only a small part of which should con preferably broiled meat, or of fish. Starches had best be adr the form of thick soups, gruels (mashed potatoes, flour, ric tender, well-cooked vegetables dressed with considerable butt visable in general not to give more than 250 or 300 grams, but permissible depends very much upon the form in which it i soups rich in extracts are usually unsuitable for patients with carbohydrates, if not fluid or also taken in other ways, may flour soup, milk soup or fruit soup. Bread, whenever possib rich in albumin (aleuronat, roborat or casein breads) or fa as bread and butter or Rademann's nutritive toast (Nährtoast) times foods containing oil (sardines in oil) may be permissible emulsion of almond oil before meals, or a not too strong fatty cheese, as, for example, the crême double de Normandie, which gourmands even eat with sugar, or with whipped cream and sugar. The effect of the latter, as has already been stated, justifies the use of sweets, such as fruit jellies, creams, sweet stewed fruits, and fruit syrups, such as raspberry syrup, grape juice, etc.—also in the form of Nectar and Pomril—as well as malt extract, malt beer, porter, and honey; in this respect the particular idiosyncrasy of the patient suffering from hyperacidity is to be considered. Among wines, as a rule, red wine is preferable, which had best be diluted with an alkaline mineral water. Among the Rhine wines, the acid varieties must be avoided; white Bordeaux wine and white Burgundy are preferable to Moselle wine. The tolerance of beer varies greatly in the individual case; in general, those rich in carbohydrates are preferable to those deficient. Coffee is usually badly borne; tea and cocoa frequently agree well with the patient, particularly if given with large quantities of cream.

Physical Treatment.—The curative factors of physical therapy from a symptomatic standpoint may aid us greatly; moist, warm compresses as well as dry heat (thermophore, Japanese stove) are particularly effective for the unpleasant sensations produced by hyperacidity. Simon maintains the favorable influence of sweating upon the gastric juice secretion, which coincides with the researches Edel made in Riegel's Clinic. I have been unable to find these so frequent as to make the method worthy of general acceptance. However, other hydrotherapeutic measures are not rarely indicated in those cases going hand in hand with neurasthenia or attributable to this.

Drug Treatment.—Drug treatment is employed in hyperacidity for various purposes. For the hyperacidity itself subcutaneous injections of atropin were advised by Riegel as valuable on account of the experiments which he made in dogs with fistula, and from his own clinical experience. Unfortunately, however, the dose required decidedly to inhibit secretion is so great that atropin can only be employed for relatively brief periods daily, unless we wish to run the risk of poisoning from atropin. For this reason atropin in doses of one milligram is particularly indicated in the treatment of acute exacerbations of hyperacidity. Whether bismuth and silver nitrate also possess the power to inhibit secretion is not certain. In systematic investigations of the gastric juice secretion prior to and after treatment with bismuth and silver nitrate lasting for several weeks, I have personally been unable to determine any absolute decrease of the gastric juice secretion. Nevertheless, these drugs at least deserve mention.

Alkalies for a long time quite properly played a rôle in the symptomatic drug treatment. It is wise to give the alkali or the alkaline mixture—usually a mixture of various alkalies is given to which, in cases complicated with constipation, a laxative salt is, as a rule, added—to the patient only on special indications, i. e., only when acidity becomes noticeable, and then not in small, divided doses but at once in a decided dose (at will cover the tip of a knife, and, if necessary, from one-half spoonful). The immediate effect of the alkaline treatment upon toms in hyperacidity proves more absolutely than any theoretic tion that a causal relation must exist between the excess of ac manifestation of the symptoms. It appears that by the admini alkalies this plus of hydrochloric acid which has a disagreeable the patient is neutralized. The prompt action of alkalies ma be explained by the assumption that a pyloric spasm and its c (painful contraction, retention of gases, etc.) are arrested by the acid, or that in hyperesthesia gastrica the acid irritation That the alkaline therapy is injurious is neither maintained by have had great experience in this treatment of hyperacidity, n observations such as have been made, for example, in the tr acidosis with large doses of an alkali. An excess of alkali is re the body with relative rapidity. In numerous original expe the administration of 10 grams of sodium bicarbonate, alkali urine usually occurred in from ten minutes to an hour and rarely lasted longer than sixteen hours. The administration to patients with hyperacidity may, under certain circumstanc crease amylolysis, which in practice is all the more striking treatment of hyperacidity the resistance of saccharifying ferm plishes but little. In association with Stargard I determined remedy last advised, takadiastase.

The treatment of hyperacidity naturally remains incompl as only the irritative condition of the gastric mucous membr bated, and not the *cause* which produces the condition. Al is not the place in which to consider this subject minutely, it must be here emphasized, above all, that in a great num of hyperacidity the simultaneous *constipation* largely demands peutic interest, for, in fact, it almost gives us the impression acidity may often be more readily cured by attacking the in the stomach. In a certain sense the relations are here inverte diarrhea of apepsia gastrica. Therefore, at least in all case acidity in which the bowel action is not perfectly normal, a Carlsbad cure—either with natural Carlsbad water or Carlsb indicated, and all the more so as in cases of hyperacidity witho tion we occasionally observe very decided improvement under t of Carlsbad salt or Carlsbad water. In every individual cas acidity we should, however, as therapeutists, reflect upon the li differential diagnosis, which in this affection are not infrequen when a case which we have believed to be "only" hyperacidi be an ulcer of the stomach, and demands of us special cons the treatment.

HYPERSECRETION

In contrast to hyperacidity, hypersecretion (parasecretion, gastrosucchorrhea, Reichmann's disease, etc.) is distinguished by a continuous "flow of gastric juice," therefore is a condition in which the stomach, even *without the stimulus of the ingesta,* secretes gastric juice. Although hypersecretion is a substantive condition, nevertheless it is not an etiologic unity, for it arises under varying circumstances. Its recognition depends upon the proof of greater or less amounts of secretion in the stomach devoid of food, and we will therefore first consider the question, what conditions, under normal circumstances, promote secretion in the empty stomach? As investigations of this point by various authors—I shall mention here Riegel, Rosin, Martius, Schreiber, Gintl and others—have furnished results in part contradictory, in answering this question I shall primarily relate my own observations. Years ago, in experiments which I made in Ewald's wards of the Augusta Hospital in Berlin, I found in 38 persons that noteworthy quantities of actual gastric juice were not present in the normal stomach, but I noted the same condition in several hundred observations which I had an opportunity of making in the course of years while introducing the stomach-tube into the stomach empty of food—partly for diagnostic, partly for therapeutic, and partly for clinico-experimental purposes. Therefore, I agree with Riegel, Ewald, and numerous other authors even more fully to-day than formerly in believing that the presence of appreciable quantities of fluid which presents all the peculiarities of gastric juice is abnormal, and that in cases in which this fluid reaches a certain amount, say exceeding 30 c.c., we have a pathologic condition.

What are the properties which lend to a fluid the *characteristics of the gastric juice?*

The fluid which usually flows in a stream from the tube is, as a rule, clear as water or turbid, of a light grey color, or may also show a greenish discoloration. The latter change is readily produced in the fluid from the fact that during the act of expression the pylorus occasionally opens and bile regurgitates from the small intestine into the stomach. In numerous observations which I had an opportunity of making, the closure of the pylorus during the act of expression is more readily overcome when fluid is present in the stomach than when it contains a thick, pappy mass. The pure secretion of the empty stomach, compared with the "residue" which represents remains of food containing chyme without a distinctly recognizable admixture of the remains of food, filters rapidly as a thin fluid, either clear as water, or occasionally slightly opalescent, or somewhat greenish (in the latter case after standing a few days the green color is frequently increased on admixture with air). If the fluid which has become turbid from admixture with leukocytes and remains of epithelia, as well as from shreds of mucus—occasionally resembling the water with which

the mouth has been cleansed—is allowed to stand, a sediment f
in more than fifty examinations of this kind I have rarely f
higher than 4 to 5 per cent. of the total fluid. This sedim
almost exclusively of mucus flocculi and nuclei of leukocytes w
plasmic ring has been digested, but also contains esophageal
epithelia which produce the turbidity. Now and then a few sta
are noted, their amount being slight, so that in a test-tube,
addition of a few drops of Lugol's solution, the sediment in pu
not show a blue color. The specific gravity of the filtrate is
abnormally low (about 1.004 to 1.008). The addition of iodin
the filtrate produces no change of color. Trommer's test is al
but the biuret test is, as a rule, positive. Upon polarization a
the left of 0.2–1.0 per cent. is shown. That true gastric juic
tain hydrochloric acid is self-evident; the values for total aci
free hydrochloric acid, however, vary greatly. The amount
hydrochloric acid is, as a rule, not very high, and the amount c
phates is usually less than in a trial breakfast. These are th
provided there is simultaneously no obvious motor insufficiency.
is not present in the "secretion from an empty stomach," we
cinæ nor sprouting yeast, and in the incubation oven the ferm
is also negative. In pure cases of the disease we find thes
immaterial whether the stomach has been thoroughly washed c
ing before or whether we have limited ourselves to prohibitin
from taking food after his supper. As Riegel emphasize
demonstrated in quite a number of cases of hypersecretion, up
ing the stomach-tube and removing the contents of the stom
vals of an hour or every two hours, we invariably observe a de
of secretion in the stomach although the patient in question
meantime, entirely abstained from alimentary stimulation.

SYMPTOMS

From this peculiarity hypersecretion is accorded a special
tion, as is its due also in a clinico-symptomatologic aspect,
symptoms which bring the patient to the physician often pre
less uniformity. The patients upon prolonged duration of th
in spite of a good appetite are usually emaciated with tissue
fluid (dry skin!), they complain of gnawing and painful sen
gastric region which are generally persistent, and—what is sy
ically worthy of remark—these are present both night and
are relieved by vomiting. The vomitus itself is usually a
acid taste, and often without admixture of food. In conse
loss of large amounts of gastric juice, there is, as a rule, th
stipation, and the urine on account of an increase of its a

quently shows a phosphate sediment (gastrogenous phosphaturia). In typical cases the test breakfast reveals certain peculiarities which are conspicuous to the expert. There is a large amount (400 c.c. and more) of very thin, fluid, stomach contents which show relatively slight sediment (the layer quotient rarely amounts to more than 20 or 30 per cent.). The sediment has an extraordinarily fine, purée-like appearance, in consequence of the excellent amylorrhexis due to prolonged contact of the gastric contents and gastric juice. The trial breakfast generally filters very rapidly, and the watery filtrate upon addition of an iodin solution (in consequence of disturbed amylolysis) usually assumes a violet or more or less bluish color. The values for total acidity and for free hydrochloric acid vary. Not rarely —but by no means always—hyperacidity is present. In several cases of hypersecretion with simultaneous disturbance in motility I have noticed the peculiar circumstance that a few hours after the ingestion of one-half to one liter of milk into a stomach which was previously washed clean, an opalescent, turbid fluid without casein flocculi (which are otherwise present but here are digested with enormous rapidity) was obtained and at the upper border of the fluid sometimes a thin, at other times a thicker, coating of cream accumulated so that a well developed intermediary layer consisting of fluid and fat was formed.

DIAGNOSIS

In the diagnosis of hypersecretion the introduction of the stomach-tube upon an empty stomach is absolutely necessary, and in the diagnostic judgment of the contents which are evacuated from the empty stomach the constituents of the secretion which have here been described are important for the reason that the "residue" of the empty stomach may easily be confounded with the "secretion" of the same. The finding in the empty stomach of an appreciable "residue" which constantly shows a higher layer quotient than that of the "secretion" from the empty stomach, is always a sign of motor insufficiency. In those cases in which we must decide the question whether pure hypersecretion or hypersecretion combined with disturbance of motility is present, I advise as the first step in the differential diagnosis the administration of a teaspoonful to a tablespoonful of currants, the stomach having been washed out the evening before, also the microscopic examination of the gastric contents for micro-parasites, finally, the fermentation test conducted in an incubation oven.[1] A minute

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[1] In the microscopic examination of the contents of the stomach in cases of hypersecretion with disturbance of motility, at a time when it should have been empty, I have occasionally found a remarkably slight amount of yeast present, and subsequently, a few times, chains of a delicate, thin, elongated variety of bacteria, as well as once distinct fungi mycelia which I also found in two cases of subacidity, and to which I attach no particular diagnostic significance.

investigation of gastric motility by the objective method of
test, and microscopic examination and fermentation in the incul
appear to me to be particularly important because in the inve
cases of hypersecretion the proofs whether a disturbance in
present or absent must be established. This differentiation is
portance for the clinical, especially for the theoretic, concepti
much more so than the otherwise justifiable classification of cas
secretion into acute or chronic, into intermittent or relapsing

PATHOGENESIS

Disputes have arisen as to the pathogenesis of hypersecr
controversy above all has revolved about the question whethe
of hypersecretion is the result of pyloric stenosis expressed
or whether it is motor insufficiency. In truth, a number of
upon hypersecretion merely as a *retention* of secretion produc
insufficiency, basing their views upon the fact that the findi
insufficiency in cases of hypersecretion is not unusual, but ev
quent. Nevertheless, various authors (Riegel, Wilkens, Ma
heim, Gintl and others) have observed undoubted cases of ch
secretion in which no objective symptoms of motor insufficie
detected, and I have reported two cases of this kind which le
upon hypersecretion as the expression of a chronic irritative
the secreting parenchyma in the etiology of which various
a part. The hypothesis that every hypersecretion is a simple
secretion, therefore, appears to me to be insufficient, because
as well as I have observed cases of motor insufficiency in whi
disturbance in motility had been removed, the symptoms of h
persisted for days and weeks before they disappeared,[1] and
series of researches which I have not as yet published I dem
contrast to Boas) that under rectal nutrition—therefore when
lus from the stomach contents was entirely absent—the amo
tion obtained from the empty stomach was just as profuse
time when food was introduced by mouth. For this reason I
nature of continuous hypersecretion to be a persistent irrit
secreting parenchyma which itself may be due to a number of
others the irritation of stagnant masses of food. However,
emphasized there is in the latter a disproportion between th
irritation and the amount of secretion, therefore a special

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[1] That such an irritation of the parenchyma may exist for some
successful gastroenterostomy I recently saw proven by the cure of a
insufficiency due to perigastritis, which should be of deep interest b
gastritis was the consequence of a laparotomy performed to recover
which had been swallowed by the patient (not while in the hospital).

the secreting parenchyma must be presupposed if the symptoms of hypersecretion become prominent. In regard to the etiologic importance of spasm of the pylorus which is occasionally observed in ulcer of the stomach —not rare in chronic hypersecretion—I limit myself to the remark that such spasms of the pylorus, under some conditions, may certainly lead to a retention of secretion in the stomach free from food, namely, if they have begun during a time in which the stomach contained no food. With the exception of ulcer of the stomach, which is frequent in hypersecretion, the pathologic anatomy of cases of hypersecretion has as yet taught us little regarding the pathogenesis of this morbid condition; for, in so far as exact investigation of the various areas of the stomach is concerned, the number of cases studied is quite scant. In a case reported by Myer and myself a few years ago there was but slight tortuosity and dissemination of the glands, also but slight change in the interstitial tissue. These findings were absent in a second case which I clinically observed. The literature, too, of the anatomical findings in cases of hypersecretion, which I cannot here describe, is contradictory. Nevertheless, in discussing the anatomy, I cannot refrain from stating that in cases of hypersecretion I have repeatedly observed gastroptosis, which, however, was usually the mechanical consequence of habitual overloading of the stomach with secretion, and also that, in those cases of hypersecretion in which an ulcer of the stomach existed, a permanent secretory irritation was perhaps due to the fact that exposed nerves upon the floor of the ulcer were irritated by the gastric contents or the gastric juice, and this, perhaps, produced inflammatory changes. Of the neurogenous type of hypersecretion those cases observed during gastric crises are the best examples. To-day, when we have learned from Pawlow's classical investigations to appreciate the relation of the nervous system to the secretion of the gastric juice, it hardly appears remarkable that hypersecretion may be of neurogenic origin. It is interesting, too, to note that hypersecretion is chiefly a disease of middle life, that it occurs more often in men than in women, and that it is more frequent in countries in which ulcer and hyperacidity are common than in other regions.

TREATMENT

The treatment of hypersecretion, primarily, is identical with the treatment of the underlying cause. Where there is a disturbance in motility, this must be combated with all the measures at our command. In those cases in which no disturbance of motility exists—which are decidedly the rarer—all the points come into question which have already been described under the treatment of hyperacidity, and here the plentiful administration of fat appears to be in order, since other authors, as well as I, have seen improvement and even cure follow its use in cases of hypersecretion. Concentrated solutions of sugar must be avoided, because the stomach in hyper-

secretion, in consequence of habitual overloading with secreti
has a tendency to myoparesis. The latter factor, in combinatio
purpose of frequently introducing acid-combining material int
ach, shows the wisdom of administering small and frequent
several reasons, those substances which increase the secretion of
juice should not be employed as food. Alkalies are advisable wh
acid secretion causes spasm of the pylorus, as in the case of h
Frequently, however, the pains are mitigated by the administrati
acid-combining substances, for example, eggs or milk. In the
the amount of albuminous food, on account of the acid-combin
ties of the albumin, should not be too small. If the palliati
just mentioned are not sufficient, amelioration may often be
the subcutaneous administration of atropin, and Reichmann
the internal administration of not too minute doses of silver
well developed cases of long duration we cannot get along
introduction of the stomach-tube, which serves a particularly
pose in evacuating the surplus amount of secretion present at
time. Lavage of the stomach with water is only necessary w
simultaneous disturbance of motility. When this becomes exp
gel advises the use of an alkaline water, and Rosenheim, as
praises irrigations with a 1–1000 solution of silver nitrate.
the measures that have been described prove inadequate, and
of the stomach for some time by rectal alimentation is ineff
gastroenterostomy come into question. In those cases in wh
no disturbance in motility this operation has a curative effect b
the discharge from the stomach of irritating and burdensom
which prevent the healing of wounds that may be present, and
ing the vomiting of food and gastric juice protects the patient
nutrition and losses of chlorin. In some cases the treatme
take into consideration the consequences of hypersecretion,
the gastroptosis, as well as the general loss or general dryne
but this is not the place in which to outline the treatment of
pathologic conditions.

In reviewing what is here stated of the diagnosis and
secretory disturbances of the stomach, the conclusion will be
the last quarter of the preceding century, in particular, has ve
added to our knowledge of these affections and broadened o
action. Our present methods have been built upon a firm
which may be more readily reviewed than was formerly the
development of an important diagnostic and therapeutic bran
pathology is due not only to a generally recognized impetus gi
cine in the second half of the preceding century, to the method
and the exact results of scientific investigations, but, abov

umen and well-directed, energetic labors of those men who have made e stomach-tube the common property of the clinic and the practising ysician (Kussmaul, v. Leube and others). The introduction of the omach-tube, and particularly the introduction of the soft stomach-tube nto medical practice—due to Ewald—was an event of almost historic nportance, as is pointed out in another article in this volume. To acnowledge this, and to express it, can nowhere be more fitting than in conluding a description of a branch of medicine in which accurate knowledge nd full comprehension have been made possible only by the use of the tomach-tube.

SUPPLEMENT

In this article on the analysis of clinical symptoms of individual secreory disturbances, it has been repeatedly intimated that an actual relation etween definite, clinical symptom-complexes and definite disturbances of ecretion cannot always be demonstrated. Therefore, it is still a mooted uestion in which cases the practitioner is justified in making an exact investigation of the gastric juice secretion by evacuating the gastric contents. This question can only be answered in a general way by the statement that a test of the gastric contents appears to be indicated in all those cases in which, without such examination, the diagnosis remains uncertain, and in which there are contraindications (in regard to this point see the article by Fleiner, pages 48 and 49) to the introduction of the gastric tube. As nothing of importance in the macroscopic investigation of the contents removed from the stomach can here be added to what was mentioned in the preceding articles, the method of *chemical* examination shall be briefly described: In each individual case we begin with dipping a piece of Congo paper in the filtered (and also in the non-filtered) gastric contents. The result of this examination indicates what further steps are to be taken in the investigation, since, according to what was said in the preceding article, if free hydrochloric acid is absent we ascertain by testing with litmus paper whether the gastric contents have an acid reaction, and, if this be the case, we examine for lactic acid. For the latter purpose I advise my modification of Uffelmann's process, since only pathologic amounts of lactic acid can be demonstrated after a test breakfast. This is carried out by using a graduated test-tube specially constructed for this purpose, which may be shaken, so that 5 c.c. of filtered gastric contents are shaken up with 20 c.c. of ether; the gastric juice and 15 c.c. of thereal extract are allowed to flow off, and the remaining 5 c.c. of ethereal xtract with 20 c.c. of water are shaken up with 2 drops of a 1 per cent. on chlorid solution (liquor ferri sesquichlorati, 1–10). If lactic acid is resent in a proportion of 5 to 1000 or more, the water turns decidedly reen. If any doubt exists in regard to the lactic acid test, it is always visable to investigate the gastric contents microscopically for an increase

of bacteria, especially the long bacteria commonly found there
cases in which we are unable to obtain much material with
tube, the latter process should never be omitted, because th
finding of numerous long bacteria in the gastric contents, a
presence or absence of the protoplasmic ring of leukocytes i
in which a chemical examination of the gastric contents car
for want of sufficient material, may give us valuable informa
ing the gastric juice secretions.

The total acidity is ascertained by the method Prof. Leo
his article (which see), and phenolphthalein is generally use
suspect apepsia this may be advantageously supplemented b
tive titration with tincture of cochineal (or with tincture of
the few cases in which we desire to determine the combine
method is advisable. If, for any reason, we wish to ascertain
tive amount of free hydrochloric acid, a few drops of a one-
alcoholic solution of dimethylamidoazobenzol is added to a m
tity (10 c.c.) of filtered gastric contents and titrated with
solution of soda until the red color disappears; or we may us
method with Congo paper. When employing the latter the
is advised for withdrawing small amounts (Ewald), and the
proceeding is to compare the drop taken from the gastric co
drop of water of the same size placed upon Congo paper. I
sier's reagent is not used from the onset (dimethylamidoaz
phenolphthalein 2.0, alcohol 100.0), after the amount of fre
acid has been obtained a few drops of an alcoholic solutio
phthalein is added, and the total acidity is determined by the

Mett's process for the quantitative estimation of pepsin
under some circumstances be of practical use in the diagno
gastrica or for the topical diagnosis of gastric carcinoma, i
out in daily practice in the following way: 10 c.c. of the filte
subacid gastric contents, to which one c.c. of a 2 per cent. solu
chloric acid which is kept on hand should be added, are plac
glass bottle with a beveled cover, or in some other low and p
wide glass vessel closed with a glass stopper. With an
prepared according to the method described upon page 92,
for 24 hours before use, and with the aid of a magnifying gl
mine the total amount of albumin digested during this tim
to my experience (and when the test is made in the manner
the normal person this rarely amounts to less than 5 mm., so
ever, to decidedly more (occasionally to double this amount
mer (*Archiv für Verdauungskrankheiten*, Bd. VIII, Heft 3 u
cently advised an ingenious modification of Mett's method of
but it would be well first to decide by comparative investiga
the additions to improve the process are absolutely necessary

tice. The dilution recently proposed by Nirenstein and Schiff (*Archiv für Verdauungskrankheiten*, Bd. VIII, Heft 6) with a 1-20th normal hydrochloric acid solution in the proportion of 1 to 16 for those cases in which we have pronounced the estimations of pepsin as valuable—cases of *extreme* subacidity—is unnecessary, as in these cases, particularly, the results of the diluted and non-diluted test appear to be almost exactly alike.

The estimation of lab-zymogen in accordance with the method proposed by Boas is best made in the following manner: 2 c.c. of gastric juice (with *high-graded* secretory insufficiency a neutralization of the acid by alkalies is only necessary when lactic acid is present and—to prevent a greater dilution—this is best done with a normal, not a decinormal, soda solution) are placed in a small measuring glass and diluted with 8 c.c. of water and 5 c.c. of milk (this must not be of acid reaction) mixed in a test-tube (dilution of 1 to 10). The remaining 5 c.c. are mixed with 5 c.c. of water so as to produce 10 c.c., and 5 c.c. of this mixture are added to 5 c.c. of milk (dilution of 1 to 20). The 5 c.c. remaining from this test are added as before to 5 c.c. of water, and one-half of this is poured off and mixed with 5 c.c. of milk (dilution of 1 to 40). By repeating this process we produce dilutions of 1 to 80, 1 to 160, and 1 to 320. After the addition to each of one c.c. of a one per cent. calcium chlorid solution, the entire series is placed in the incubation oven, heated to 37° C., and allowed to remain for half an hour. Under these conditions the filtrate from a stomach which functions normally will coagulate milk in a dilution of 1 to 80 or even 1 to 160. Glässner (*Berliner klin. Wochenschr.*, 1902, Nr. 29), reports that the normal amount of lab-ferment in the gastric juice obtained by a test breakfast is 1 to 100, i. e., 0.1 c.c. of neutralized gastric juice will, within half an hour, caseate 10 c.c. of milk at a temperature of 30° to 40° C.

For further details of the methods of examination, the reader is referred to recent text-books upon the diagnosis and therapy of gastric diseases, especially to those of Boas, Ewald, Fleiner, Riegel, and Rosenheim, as well as to the text-books upon microscopico-chemical diagnosis by v. Jaksch, Lenhartz and others. A critical compilation of the literature up to the year 1892 concerning the various methods of examination of the gastric juice is found in the monograph by Martius-Lüttke: "Die Magensäure des Menschen," Stuttgart, F. Enke, 1892.

DIAGNOSIS AND TREATMENT OF GA DILATATION

By F. RIEGEL, Giessen

CLINICAL FORMS

When a patient presents himself to us and states that he frequent vomiting of large residua of food, and that he rep remains of food that he had consumed several days before, examination of the patient, the lower boundary of the stom below the umbilicus with an approximate normal position o and when, throughout this entire region, a loud succussion s produced, the suspicion of dilatation of the stomach is obv such a case, seven hours after a test-meal, profuse quantitie more, are evacuated from the organ, and if an evacuation in upon a presumably empty stomach shows profuse remains, *gastric dilatation,* or *gastrectasis.*

Two factors are necessary for the diagnosis of gastric *decided increase in the size of the organ* and an *insufficient m* a *so-called motor insufficiency.*

I do not agree with those modern investigators who rece peatedly declared that the terms "ectasis" and "dilatation of t are no longer expressive of present scientific views, if they in inate the term "gastric dilatation" from pathology, and t place "mechanical insufficiency, myasthenia gastrica, ichoch gastric debility" or the like, which denote exclusively insuf activity. These authors are certainly correct when they dec size of the stomach is no measure of its motor activity. A be larger than normal, yet, notwithstanding, have sufficient and another stomach may be well within the usual physiolog nevertheless, show motor insufficiency. Boas is, therefore, ce when he says that we drift from a positive foundation in diagn make the limits and size of the stomach a criterion in deci existing gastric dilatation. But no conscientious physician diagnosticate gastric dilatation upon the basis alone of a large the gastric limits.

The stomach may have suffered in its elasticity, may be r ble, yet, nevertheless, still be capable of propelling its contents time. If such a stomach be inflated with air or carbonic acid, it reaches far beyond the normal limits; such a stomach is abnormally elastic; nevertheless, it may be able to propel the ingesta at the right time like a normal stomach. This condition is designated *megalogastria.*

Such cases are discovered purely by accident; for megalogastria, as such, does not give rise to symptoms. The conception of ectasis demands more than an increase of size, more than abnormal elasticity; above all, it is necessary that the stomach should be unable to propel its contents at the proper time, that during the long period in which a normal stomach receives no food, more or less profuse contents are still within its walls.

We are forced to admit that the designation "gastric dilatation" primarily implies only a larger extent of space. Nevertheless, this same term "dilatation," in a certain sense, also indicates a condition of motor insufficiency, for, from what other cause has the patient with gastrectasis a dilated stomach, wherein is the root of his difficulties except in this, that the stomach is incapable of propelling its contents, and is, therefore, constantly overfilled! Even the older physicians in their conception of gastric dilatation not only held the view of an abnormally large extension, but also that the stomach lacked the power of timely propulsion of its contents. That stomach alone is *permanently* dilated which cannot empty itself at normal periods.

If this view of gastric dilatation be accepted—and formerly it was universal—there is absolutely no reason for discarding the old, established designation "gastric dilatation." It is true that the determination of the size of the stomach alone is not sufficient, it is also necessary to ascertain the motor activity of the dilated organ. At the bedside it is never satisfactory to determine the physical changes only of the diseased organ; we must also picture to ourselves the disturbed function.

Similarly, we speak of a dilatation of the heart; but as, in the heart, the determination merely of an increase in size is not sufficient, so, in every case, we study the effect of this dilatation upon the circulation, both in the arterial and venous system, since increase in size is by no means always of like importance; this is also true of the stomach. In the conception of dilatation, a decrease in the motor power is included as well as increase of size. At the onset, however, it is clear that it makes a decided difference whether a stomach shows only decreased motor power, or whether it simultaneously shows a decided and permanent increase in size.

It must be admitted that formerly the error was frequently made of diagnosticating gastric dilatation solely because of an increase in size. But to-day no conscientious physician, upon suspicion of gastric dilatation, will limit himself only to determining the boundaries of the stomach. This would be just as one-sided and wrong as to make a diagnosis of gastric disease from the determination of the acidity alone, or to diagnosticate carcinoma from the absence of free hydrochloric acid alone. *The increase*

in size primarily awakens the suspicion of dilatation, but thi
cient warrant for a diagnosis.

When there is a decided distention of the stomach combine
jective symptoms, the physician should not neglect to introduce t
tube, to determine the functions of the stomach in regard to
secretion.

The name *"motor insufficiency"* indicates only a *disturbai*
tion which may occur in the most varied diseases, and is ma
the gastric contents are not expelled at the proper time. Thi
in a dilated stomach, in a normal one, and even in a stomach
size. The designation "motor insufficiency" in place of "dila
not be regarded as correct, for the reason that motor insuffic
ble without any enlargement of the stomach. It is, however,
consequence, whether, in the given case, the motor insufficie
to a permanent abnormal distention of the stomach or not, but
latter case do we speak of *dilatation.*

Atony is a special form of motor insufficiency. By this
condition in which the tonus of the gastric musculature is di
consequence of which there is delay in propelling its contents,
boundaries of the stomach having exceeded their normal li
may lead to dilatation, but need not necessarily. When the
case, we speak of *atonic dilatation.* The atonic stomach propel
contents more slowly than is normal, but does finally propel t

We must, therefore, differentiate between motor *insufficien*
special variety of motor insufficiency), and *dilatation.* Megal
not here come into question.

Before proceeding to the clinical picture of dilatation—an
speak of the clinical picture of dilatation, and not of the disea
for it is not a disease, *sui generis*—I thought it necessary t
conception of the term. On this point, as a glance at the
recent years will show, there is a great difference of opinion.[1]

An attempt has been made to substitute a different design
term in general use, "dilatation." If the conception "gastri
is considered in the above mentioned sense, there is no re
introduction of new terms, as they have no advantage over the
ally, in determining a dilatation we must not limit ourselves
estimation of the size, but we must endeavor to investigate
of the disturbance in function in connection therewith.

There are varying *degrees* in the nature of the clinical
we designate by the term "ectasis," but no well-defined limi
we may speak of dilatation and from which we should sp

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[1] I refer particularly to Hesse, "The Conception of, and the Term, '
tion,' in German Literature since 1875." *Berliner klin. Wochenschr*
and 24.

insufficiency and atony. The diagnosis of gastric dilatation is easy when the stomach permanently contains decided residue of food, and when its boundaries, therefore, far exceed the normal limits. In milder cases the condition is different.

No stomach, not even a normal one, *constantly* shows the same distention; its boundaries vary, we may almost say hourly. The same is true of the dilated stomach, and also of the stomach with motor insufficiency, which, although it finally expels the ingesta, does this decidedly more slowly than the normal organ. The stomach with motor insufficiency is *relatively* dilated in so far that it still retains ingesta at a time when a normal stomach has already expelled it.

In this sense we speak of *relative dilatation* if a stomach, seven hours after a test-meal, still contains considerable remains of food, while its limits do not exceed the normal boundaries. For, at this time, the stomach should be empty and markedly contracted. In such cases we do not make a diagnosis of gastric dilatation, but only when the lower and lateral borders of the stomach permanently exceed the normal boundaries, and when the stomach *permanently* retains ingesta. In spite of this, in the first mentioned case, there is a relative and *temporary* increase in size, and there can be no doubt that, upon prolonged duration and long-continued action of these deleterious causes in such a dilated stomach, true dilatation may gradually develop.

It is, therefore, evident that there is no sharp dividing line between dilatation and motor insufficiency. Dilatation naturally includes motor insufficiency, for the stomach can only be dilated when its contents are not entirely expelled at the right time. Every true dilatation, therefore, must be accompanied by motor insufficiency; but not every insufficiency need lead to true ectasis. It is obvious, therefore, that in discussing dilatation we also consider motor insufficiency and atony.

As to the *causes* of dilatation, it must be remembered that this can only develop upon a basis of motor insufficiency. If the stomach cannot discharge its contents fully and at the proper time, it must be distended longer than is normal, it must remain abnormally weighted. Gradually, decided distention occurs, and, finally, true ectasis.

Motor insufficiency can only arise *where there is a disproportion between requisite labor and the capacity for labor, where requisite labor and power of labor no longer, as in the norm, maintain an equilibrium.* When the motor power of the stomach is no longer commensurate with the demands made upon it, the work is only imperfectly done or after a long period; i. e., the stomach will expel the ingesta only partially or after a prolonged period of time. The causes of this diminished motor activity are various, and may be expressed as follows: 1. The amount to be propelled is abnormally large, as, for example, in acute, or prolonged, or frequently repeated over-distention of the stomach; 2. The expelling power, the muscular power of the stomach, is deficient; 3. There is an abnormal re expulsion of the ingesta from the stomach.

While, in the group first mentioned, we are dealing with age or diminution of the motor power, in the third group, th ical hindrance to the expulsion of the ingesta, we often find power increased, the muscularis even hypertrophic the same of a valve of the heart, that portion of the heart anterior t is not only dilated but, at the same time, it becomes hypertrc theless, it is incapable of increased labor. This group rep the greatest number of dilatations, and includes the *stenoses and its surroundings.*

Much rarer is insufficiency, the *atony and atonic dilata* hand in hand with flaccidity of the muscularis. Moderate de are not rare. Such an atony may arise when the stomach eith or for a long time has an unusual amount of work to perfo see atony as an accompanying symptom of various affections We observe it particularly after acute distention of the ston difficult to digest, we see it in persons who eat hurriedly a and chew their food insufficiently. True dilatation may quence of such over-distention; *in children,* particularly, *a* not rarely occurs in this manner. Occasionally it increases and not seldom such acute dilatations have a lethal outcome. case, other factors besides an acute distention of the stoma rôle. Thus Kussmaul[1] many years ago called attention t in gastric dilatation all the symptoms of complete closure m appear and continue until large amounts of the stomach c pelled by vomiting or by lavage.

As Kussmaul pointed out, such cases cannot be due to the muscular wall by immoderate distention, for peristalsis is seen to be markedly active when the stomach contents ca propelled. Kussmaul believes that in cases of this kind a tug and tension occurs at the point where the horizontal duodenum leads into the vertically fixed part at the verteb

This conception of the combined action of a mechanical gained decided support by the recent investigations of All ing "Arterio-Mesenteric Intestinal Occlusion at the Du Boundary and its Causal Relation to Gastrectasis." Albr that the cases of acute gastric dilatation, above all with sy (intestinal obstruction), often have their origin in an ar invagination of the inferior transverse portion of the duode stomach, as it dilates more and more, forces the transvers loops of the small intestine downward toward the pelvis, the mesentery, and particularly the arched fold under which the duodenum enters into the jejunum, becomes tense, and thus a constriction is caused in the mesenteric artery at the duodeno-jejunal boundary. Albrecht quite properly states that the vicious circle which is formed by the downward sinking of the folds of the small intestine causes tension in the mesentery which completes the compression of the duodenum. Thus the contents of the duodenum become more and more engorged, and the stomach constantly more full and dilated. The horizontal portion of the duodenum becomes still more overloaded, and the downward displacement of the colon and the small intestine still further increased.

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[1] "Peristaltic Unrest of the Stomach." *Volkmann's Samml. kli*

[2] *Virchow's Archiv,* CLVI.

That an acute atony of the stomach, an acute gastric dilatation, may favor the development to a high degree of an arterio-mesenteric duodenal incarceration is clear. In other cases, the compression of the duodenum by the mesentery of the small intestine may have been the beginning of the disastrous chain. In incomplete closure, and with an incomplete dislocation of the duodenum, there may be chronic dilatation with vomiting of bilious masses.

In cases of this kind recently reported, the symptom-complex was observed after chloroform narcosis preceding various operations, among them some upon the biliary passages. Bäumler,[1] in reports of two cases belonging to this category, has recently stated that numerous factors favor this form of intestinal immobility, and he groups these as follows: 1. Greater length of the mesentery of the small intestine; 2. Gastroptosis; 3. Enteroptosis in its influence upon the position of the duodenum and upon the root of the mesentery; 4. The position of the duodeno-jejunal boundary in relation to the vertebral column; 5. Marked lumbar lordosis; 6. High-graded emaciation and debility from preceding disease; 7. Chronic gastric dilatation in consequence of congenital or acquired stenosis of the pylorus; 8. Prolonged dorsal decubitus after operations; 9. Very complete emptying of the intestine prior to an operation.

Further predisposing factors for the development of atony in general are preceding severe infectious diseases, general constitutional affections, anemia, chlorosis, and the like.

Those who are poorly nourished, or debilitated by sexual excess or other causes, are highly susceptible to atony. Abnormal position of the stomach, gastroptosis, enteroptosis, extreme flaccidity of the abdominal walls may be designated as predisposing factors, even although they do not in themselves cause atony.

In some cases, the so-called weak, atonic stomach is *hereditary*. Frequently, the nutrition habitual from childhood may here play a rôle. Like every muscular organ, the stomach, from childhood, should be accustomed to proper food, as this develops its muscular activity. M(which is deficient in residue may so weaken the stomach tha unusual circumstances, demands are made upon it which designated as harmful, its muscular tonus suffers and react flaccid.

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[1] *Münchener med. Wochenschr.* 1901, Nr. 17.

At the onset, we might suppose that disturbances in tl the gastric juice would decidedly influence the propulsion contents, and thus indirectly cause disturbances of motor acti observation shows that this is not the case to the extent, and that we might be inclined to assume. Such a reaction we sh for in pure *achylia* in which the secretion of gastric juice is (But here, particularly, such a disturbance does not appeai trary, the motor power is good throughout, and even incre best explained by studying the remarkable results of the Pawlow[1] in which he proved that the duodenal mucous mem the propulsion of the gastric contents into the intestine, re condition of fulness in the latter, from the fact that it dej reaction of the gastric contents and their acidity. Alkalin(ally pass from the stomach very soon. On the other hand, tine has received a portion of the acid gastric contents, it c action a temporary closure of the pylorus. The acid food n pylorus has permitted to pass through, causes an increas(pancreatic juice, and by and by becomes neutralized. Onl) occurred is the expulsion of further acid masses from th(mitted, and, accordingly, the quickened motor activity of achylia is readily understood.

On the other hand, in cases of increased gastric juice ticularly in *hypersecretion,* we not infrequently see distu motor activity in the form of delayed expulsion of the ir rarely also of ectasis. The connection between these is Many authors regard hypersecretion in all cases as secondar quence, not the cause, of ectasis. That hypersecretion and quently observed in combination is certain; which is prim secondary, cannot be so readily decided. Even where an stenosis can be determined, this does not prove that the mec tion is the cause of the hypersecretion. Pyloric stenosis in a sufficient secretion of gastric juice by no means always secretion. That hypersecretion often rapidly disappear enterostomy, which so greatly facilitates the flow of the s(be regarded as a proof that the latter is a consequence of t longed stasis may keep up the irritative condition, its dis emove it, and facilitate the outpouring of the secretion; but stenosis in itself does not explain the prolonged secretion of gastric juice.

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[1] "Das experiment als zeitgemässe und einheitliche Methode n schung." Wiesbaden, 1900.

The secretion of gastric juice normally is not continuous, but periodic. As Pawlow[1] first showed by conclusive experiments, the normal stomach, empty of food, does not secrete gastric juice. It only secretes it upon *digestive*, not upon mechanical, stimulation. For this reason, therefore, it is not to be expected that stenosis in itself will cause a continuous secretion of gastric juice. If the stomach of a patient suffering from hypersecretion is washed perfectly clean, and is evacuated after a few hours, during which time the patient has taken no food, a greater or less amount of secretion is nevertheless found. The stomach therefore, despite the absence of any digestive stimulation, has spontaneously secreted gastric juice. This certainly favors abnormal stimulation. On the other hand, we also see, although not so frequently, cases of hypersecretion without pyloric stenosis and without actual dilatation. It is true these cases are readily overlooked. Hence it follows that pyloric stenosis in itself is not the primal, and not the only, cause of hypersecretion. Inversely, however, it is doubtless true that hypersecretion alone may lead to dilatation by limiting the transformation of the amylacea to such a degree that upon prolonged retention fermentation of gas is produced, and, finally, if this continue for some time causes dilatation. According to Pawlow's investigations, the abnormally acid contents and the profuse amount of acid-secretion remain in the stomach a much longer time than the gastric contents which are normally acid. The transformation of the acid gastric digestion to the alkaline intestinal digestion by the pancreatic juice must be slow and difficult in those cases in which a much higher degree of acidity and a much larger amount of acid gastric juice is present than in the normal, as is the case in hypersecretion.

Particularly in the case of hypersecretion, spastic processes at the pylorus are observed which inhibit the exit of the acid ingesta; this, accordingly, can only be regarded as a proper preventive, as a protection against a disturbance of digestion of the small intestine. These factors are calculated to produce a long stagnation of the ingesta even without the existence of a stenosis. Hypersecretion in itself must cause stasis, and may, therefore, in itself finally produce dilatation. Hence it cannot appear strange that, particularly in the case of hypersecretion, a change in the symptoms is observed, sometimes improvement, sometimes aggravation, sometimes an increase of the motor insufficiency.

It has been maintained that marked *psychical* influences give rise to such atonies. That psychical influences may affect the secretion of the gastric juice and also the motility of the stomach is no doubt true. But these are only transitory disturbances. It has not yet been proven that chronic ectasis may develop upon a purely nervous basis.

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[1] "Die Arbeit der Verdauungsdrüsen," 1898.

Whether, and in how far, diseases of the *central nervous s* to atony and dilatation has not yet been determined with certa lated, recent observations appear to favor the possibility of tl ment of gastric dilatation from central influences.

The cases are not rare in which patients with gastrectasis beginning of their disturbances to a *single trauma* in the gast As is shown by careful analysis of these cases, many of the caused by a stenosis of the pylorus or in its vicinity. This st number of cases originates in a cicatricial narrowing brough necrosis and ulceration due to trauma; in other cases the t causes a perigastritis, and, in consequence of this, adhesions to th ing organs; when these adhesions in their further course affect of the pylorus, they also lead to stenosis. These traumatic st the peculiarity that the stenotic symptoms never develop at often only a long time after the trauma.

Simple *atony* may also develop after trauma; it has been operations upon the abdomen. In contrast with the form just atonies after traumata mostly develop suddenly and soon after they may even themselves prove fatal, as in a case recently Grundzach.[1]

Not only a single trauma, but also *frequently repeated ones* the gastric region play a rôle in the etiology of ectasis.

Many occupations necessitate that the labor be performed i which causes the body to be bent forward, that the epigastrium to the pressure of tools which are stemmed against it, and it to long-continued pressure. This may cause circulatory di pressure ulcer, but also cicatricial stenoses or perigastritis, a to gastrectasis. But we cannot assume a cicatricial stenosis i of gastrectasis; even active peristalsis of the stomach is no p At all events, in a number of dilatations developed in this note decided variation in the symptoms, and, under some c even complete retardation, so that the assumption that sp pylorus are chiefly the cause does not appear unjustifiable.

It has been maintained that some *narcotics*, such as alcohol have a relaxing and paralyzing effect upon the muscularis of No cases developing in this manner have as yet been dete certainty.

Although it cannot be denied that gastrectasis may deve basis of atony, nevertheless, at the onset, in every dilatatio degree the suspicion is justified that a mechanical hindrance, stenosis either at the pylorus or in its vicinity, is the cause tion. *Mechanical hindrances are by far the most frequent cau* and the most important of these are *cicatrices from ulcers, carcinoma,* and *cicatrices from caustics.*

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[1] *Revue de Médecine*, Mars, 1899.

The consequences of dilatation will naturally also appear when the obstruction which causes stenosis is situated somewhat lower down, below the pylorus and in the beginning of the duodenum. *Foreign bodies* which find their way into the pylorus, cherry stones, medlar stones, invaginated gall-stones, have caused dilatation there or in the duodenum. Quite exceptionally, cases of tuberculous stenosis of the pylorus have been observed. Jacobi [1] has recently reported such a case. Very rare causes of pyloric stenosis are benign tumors, pediculated polypi, connective tissue hypertrophy of the pylorus, external tumors pressing upon the pylorus (large gall-stones in the gall-bladder, Minkowski).

Pyloric stenosis is rarely *congenital;* nevertheless, quite a series of reports are at hand in which a decided hypertrophy of the pylorus caused marked symptoms of stenosis in nurslings. It is true a number of cases of supposed congenital pyloric stenosis admit of a different explanation. Pfaundler,[2] particularly, has called attention to the fact that recently in examining cadavers of nurslings, in about every third case the stomach was found in a peculiar state of persistent, muscular rigidity. The pylorus itself, in consequence of the permanent contraction, was very narrow, and frequently did not permit the passage of a medium thick sound. Pfaundler believes that many cases reported as congenital pyloric stenosis correspond minutely with the findings in systolic stomachs of this kind. However, he does not deny that there are actual clinical pictures among nurslings which point to an existing pyloric stenosis.

These are the most important causes of dilatation and motor insufficiency. The *degree* is a varying one. High-graded ectasis is observed in organic stenosis, among which by far the most frequent causes are carcinoma and cicatrices from ulcers.

PATHOLOGY

Anatomically, the picture presented by ectasis is in proportion to the cause and duration of the dilatation, the degree and nature of the stenosis. Motor insufficiency, as such, so long as it does not give rise to dilatation, is not susceptible to anatomical diagnosis, or, at most, only in the rare cases with cirrhotic change or diffuse malignant degeneration of the stomach.

The degree of dilatation varies; primarily, and chiefly, the fundus and the greater curvature which bear the brunt of the affection are involved in the dilatation. But, in severe grades, the dilatation is more general, chiefly lateral, as may often be determined *intra vitam.*

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[1] *Wiener klinische Wochenschrift,* 1900, Nr. 48.

[2] *Ibid.,* 1898, Nr. 52.

In isolated cases of ectasis the walls of the stomach are very affected; sometimes they are thinned, in other cases they sh thickening. This variation depends primarily upon the cause tation, and, secondly, upon the duration of the affection.

Decided hypertrophy, particularly of the pylorus, is seen stenoses of the pylorus and of its surrounding areas. The m often three or four times thicker than the norm. In other ca no hypertrophy of this kind; on the contrary, the muscularis a and atrophic as in the case of atonic dilatation; not infrequen and fatty and other forms of degeneration of the muscularis in the later stages of long-existing ectasis.

The relation of the sub-mucosa and mucosa depends much le dilatation itself than upon the underlying causes of the ectas regard to these points we must refer the reader to the indiv of disease which produce dilatation.

SYMPTOMATOLOGY

In the description of the clinical picture, "ectasis," I shall entirely to the symtomatology of dilatation. It would be digression if I were to consider all varieties which might secon dilatation, such as carcinoma, cicatrices from ulcers, perigastr chronic gastritis, hypersecretion and the like. Here we will c the clinical picture of dilatation as we meet it in daily practice the diagnosis, "ectasis," is an imperfect one; it tells us o stomach is dilated, and that motor insufficiency is present; our be to determine the cause, and, as a rule, this offers no ins difficulty.

The cases are comparatively rare in which the special dia cause of the dilatation is difficult. Often the examination o not only suffices for this but enables us to determine the und tion which produced it. Naturally, there are cases in whi observation and the most minute investigation of the course and of the results of treatment are necessary; but at the bedsi first determine the dilatation, and then seek for its cause.

The symptomatology naturally varies with the degree of t Mild grades of ectasis produce less prominent symptoms than As I have already stated, there is no sharp division between ciency and dilatation, in spite of many attempts to define it. ble to draw a line, from which point the stomach may b dilated. This might be feasible in an organ always of absolu size, but not in an organ which, like the stomach, shows a v sion at different periods. Naturally there are extreme degree which enable us to say, without more ado, that the stomac dilated. Slighter degrees are designated by one author only as motor insufficiency, by another, as dilatation. In estimating the size of the stomach, we must consider the period as well as the absolute degree of distention.

In no group of diseases is the *time of the physical examination* so important as in gastric affections. It is by no means immaterial at what time of the day we examine a patient suffering from gastric disease. If, early in the morning before food is taken, we can elicit a distinct splashing sound, and so prove that large quantities of fluid and air are still in the stomach when it should be empty, we decide at once that motor insufficiency is present. In a certain sense we may also consider this stomach to be dilated although its lower boundary does not extend below the umbilicus. For it is at least temporarily dilated; i. e., more than normally distended; early in the morning it should be empty and contracted. We may, therefore, speak of relative dilatation in contrast to absolute and extreme dilatation. In practice, however, it is necessary not only to determine the extreme grades, but also the slighter forms of dilatation and motor insufficiency.

The earlier the stage in which the diagnosis is made the greater the chances of controlling the malady. But, as I should like to emphasize at this point, to obtain a clear insight we must employ all methods in the examination. A well-trained practitioner will sometimes, on first observing the abdomen, be able to diagnosticate gastric dilatation just as we diagnosticate tabes dorsalis from the gait, and aortic insufficiency from the pulse. But no conscientious physician would be content with this. For the name, alone, of the disease, does not suffice, but, at the bedside, we must fully comprehend the nature and intensity of the physiologic disturbance of function, and for this purpose we require numerous methods of investigation.

If this had always been borne in mind in making the diagnosis of ectasis, we should not have erred by regarding the size of the stomach as of most importance, and of considering this alone the decisive factor. But it is just as partial and extreme to determine the degree of ectasis from the amount of material evacuated or from the amount of residue, and to consider the size of the organ as entirely secondary. They are related to each other, and still further methods of investigation must be resorted to if certainty in diagnosis is to be assured, as well as a full understanding of the nature and intensity of the disturbances in a given case.

To begin with the **subjective symptoms**, it must be remarked that these vary not alone according to the degree of the dilatation, but also according to the cause and the manner of its development. As a rule the first symptom of which the patients complain is a feeling of tension, fulness, and pressure after meals. Some experience abnormal sensations of this kind even after light meals; some report that they feel better in the recum-

bent posture than when standing, particularly after a somew
meal.

Others complain of eructations and pyrosis, others again of
of active unrest in the stomach which sometimes increase
spasmodic pain. Occasionally these disturbances are relieved
This is observed chiefly in pyloric stenosis with sufficient or inc
tion of gastric juice; but spastic conditions of the pyloric ri
of themselves produce painful spasmodic attacks.

The *appetite* is capricious; as a rule it is decreased; on
mild cases is it normal or increased. Often the patients have
satiety after partaking of small amounts of food; bulimia oc
oxysmally is rarely complained of.

The *thirst* is more important than the state of the appetite
all well marked cases there is complaint of thirst. The more
degree of dilatation and motor insufficiency, the more mark
increased. The stomach itself does not absorb water, but, on
a more or less active excretion of water occurs in the stom
absorption of many substances, such as sugar, dextrin, alco
etc.; with well marked dilatation of the stomach the propulsio
comes exceedingly difficult. This remarkable increase in thi
explained; and the fact that in dilatation more fluid is freq
in the stomach than is consumed is comprehensible. The dec
tion of water corresponds with the decreased excretion of ur
we shall recur in describing the objective symptoms.

The state of the *tongue* is not characteristic; in the more
there is a noteworthy tendency to dryness. In general, tl
of the tongue varies; but it is by no means, as was formerl
reflection of the stomach.

An important and, probably, the most important sympton
is *vomiting*. In the milder grades of motor insufficiency i
tirely absent; in well marked ectasis it is almost always pres
in varying frequency. In some cases its peculiarities at onc
tation. Thus, if the quantity of the vomited material excee
of the last meal, if remains of food which had been taken pr
some days before, be detected in the vomitus, motor insu
dicated.

The patients are usually decidedly relieved by vomiti
reason they often attempt to produce it voluntarily. Von
repeated daily; frequently it occurs at very irregular interv

The appearance of the vomited material varies greatly;
depend chiefly upon the underlying affection which causes
and upon the secretion of gastric juice. The vomitus in
hypersecretion has a different appearance from that due t
the pylorus. In the former case we observe the well know

the lower layer consisting of fine remains of starch, the next of a decidedly acid fluid, and the upper layer being frothy as the expression of gas fermentation; in the latter case coarse remains of food are seen, particularly remains of meat, with an absence of free hydrochloric acid, and a decided reaction for lactic acid. I cannot here describe the special peculiarities of the vomited material, which differ according to the underlying affection that causes the dilatation. I must refer the reader to the individual diseases themselves.

An admixture of blood in the vomitus is not rare; this is most frequently seen in carcinoma, but it also occurs in other processes. Naturally this finding has nothing to do with the dilatation as such. Moderate degrees of motor insufficiency often exist for a long time without impairing the general condition. Patients with high-graded ectasis, however, as a rule, are feeble, weak, and incapable of exertion. We commonly meet with complaints of numbness in the head, of headache and of vertigo.

The *bowels*, as a rule, are constipated; occasionally not. Very rarely do the patients complain of diarrhea; this is most frequently observed in stenoses with marked gaseous fermentation.

These most important symptoms of the patients, as will be noted, show but slight peculiarities. The **objective symptoms** are much more important, and are alone decisive in diagnosis. Naturally they also differ according to the degree of the dilatation. Slight grades of atony and motor insufficiency at first, that is, if the duration has not been too prolonged, do not materially affect the *general condition and nutrition*. It is different in high-graded, long-continued dilatation. Here the nutrition suffers decidedly. The patients are more or less emaciated; the fatty layer has disappeared, the skin is dry and withered, it may be raised in large folds and desquamates; the muscles are thin and atrophic. This picture is met with not only in cases of dilatation due to carcinoma, but is also observed in dilatation occurring in benign affections, and, naturally, it depends upon the duration of the disease. Carcinomatous dilatation leads more rapidly to loss of strength than the benign forms. But, in any given case, the appearance, the degree of emaciation, cannot be taken as positive indications of the nature of the stenosis. Any one who has seen many patients of this kind will admit that the external habitus is not decisive. The nature of the stenosis can only be determined by a study of the entire condition.

The examination of the diseased organ must be made systematically, and, in every case of gastric disease, should begin *with inspection*. I expressly emphasize this method of examination because in practice it is often neglected. As, in the examination of the heart, the sequence of the investigation is essential, so is it in the stomach. Inspection often gives us important knowledge; naturally, not in very fat persons. On the other hand, in slender persons, poor in fat, with thin flaccid abdominal walls,

we often see the contours of the protruding and dilated stoma
other aid. This, of course, depends upon the *time* of the exami
we examine immediately after the stomach has rid itself of
part of its contents by vomiting or by lavage, inspection usu
nothing. If the stomach, however, is still quite full, we can
trace its boundaries; the upper portion of the epigastrium, p
somewhat sunken, is seen lower down extending far below the n
a protuberance is noted, inside of which there is often active
movement from left to right. Upon superficial investigation th
tic movements may perhaps convey the impression of being ir
closer examination, however, a certain regularity is observed,
tions and depressions may be recognized. The boundaries of
are not only sharply defined, but at the same time we concl
are not dealing with a simple flaccidity. In atony such activ
movements are never observed. By inspection alone, we can,
circumstances, determine the presence of an ectasis, but also sir
that the stomach labors with *increased force*. The latter c
favors *stenosis*; of course, this need not be an organic obstructi
a functional disturbance, a spasmodic contraction of the pyloru
porarily make the outflow difficult, and produce an increase o

For a positive diagnosis of dilatation it is absolutely
observe accurately not only the lower but also the *upper* boun
well known, marked dilatations frequently, even as a rule,
gastroptosis, i. e., the whole stomach is displaced downward.
tion of the lower border alone to a point more or less below th
not prove dilatation, for this also happens in gastroptosis; t
upper boundary must be accurately located. Normally this
if it be displaced downward, we note in the epigastrium a s
ing, and below this a prominence gradually increasing as it d
with its convexity directed downward.

I shall not here discuss the results of *electric illumina*
from the fact that this method can only be utilized in isolat
it gives no results which may not be obtained in other and sim

Inspection is to be followed by *palpation*. By this mean
the results of inspection and also gain other valuable knowled
patients, provided the stomach is not empty, we may often d
pate the organ under the flaccid abdominal walls, particularl
is laid flat with the ulnar surface next the abdomen, and, lig
glides downward. The practised hand frequently traces th
of the stomach accurately. Simultaneously the proof of a
pyloric region sometimes complements the diagnosis of such

Less importance is to be attached to the method formerly
tised—of *palpation by means of a sound*. Although it is
frequently succeed in feeling the end of the sound through t

walls, we find at most only the lower boundary of the stomach, and nothing showing its total extension; moreover, a bend in the sound may very readily lead to errors.

Another symptom that may be determined by palpation is the *splashing sound* which we elicit by tapping the abdominal coverings with the finger. Splashing sounds only permit diagnostic conclusions when we *simultaneously consider the time and the extent of their development.* In themselves they only show the presence of fluid and air in a hollow organ. Splashing sounds may, under some circumstances, also be produced in healthy persons, but only when the stomach contains air and fluid, and even when this is the case not beyond the normal boundaries of the stomach. If we find splashing sounds extending beyond these, if we find them within abnormal limits at a time when a healthy stomach should be empty, that is, seven hours after a meal or in the morning before food is taken, diagnostic significance may be attached to them.

Such a splashing sound proves that the stomach still contains more or less profuse amounts of fluid, and it further proves that the stomach is more decidedly dilated than the normal. Naturally, if the latter conclusion is justified, gastroptosis must be excluded.

To determine the boundaries and position of the stomach it is best to *inflate it by means of air or carbonic acid,* and the choice between these two methods has been much discussed. We would digress too far to enumerate all the objections which have been made against inflation by carbonic acid. I believe, however, that these objections are entirely unfounded. I have never seen deleterious effects, or even danger from it, although I have used this method daily for many years. But doses sufficiently large must be given, a heaping teaspoonful of sodium bicarbonate and a correspondingly large dose of tartaric acid; less than this is insufficient.

Distention by means of bellows, and autodistention, such as has lately been advised by Spirack[1] have the disadvantage that a stomach-tube is necessary. The administration of the effervescing mixture is easier and more readily carried out. If the abdominal walls are not too tense and fat we usually succeed, without more ado, in tracing the contours of the stomach; where this is impossible, its boundaries are frequently revealed by the uniformly elastic sensation and by percussion. In stenosis of the pylorus, after the administration of an effervescing mixture, the peristaltic movements of the stomach usually show a marked increase in activity.

Percussion of the stomach serves to confirm and emphasize the previously mentioned results; primarily it only aids us in tracing the boundaries of the stomach, therefore in *gauging its size,* but does not prove a simultaneously existing motor insufficiency. But it does confirm the latter if we succeed in ascertaining the presence of fluid in the a time when it should be empty. For this purpose it is examine the patient while in the erect and the recumbent pc nately. If, when standing, we percuss downward, and if the of the stomach are first filled, we find a tympanitic zone whic at the height of the navel, or somewhat below or even some by a horizontal dull line, which lower down terminates in a convex curve, but which in the recumbent posture again cl guard against errors, it is advisable in such cases to evacuate and thus control it. An evacuation of this kind is, *eo ipso,* all cases where there is the slightest suspicion of disturbance activity of the stomach.

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[1] *Deutsche med. Wochenschr.*, 1900, Nr. 23.

The Penzoldt-Dehio method, which consists in administ patient early in the morning upon an empty stomach two or of water in succession, and determining after each glass the gastric dulness while the patient stands erect, is more useful ing the lower border of the stomach and its tonus than for dilatation. It may be dispensed with in cases of high-grad which the stomach is never entirely empty. Only in mild gr insufficiency is it worthy of trial, and even here it is superf diagnostic evacuation will give us much more positive proof ence of motor insufficiency, and, at the same time, acquaint degree of the same.

Some of the other numerous methods for determining capacity of the stomach are very complicated, have no ad those just mentioned, and, for this reason, may be ignored is true of the method recently proposed by Queirolo.[1]

If the methods of examination described have given an the existence of a dilatation of motor insufficiency of mil produce a *diagnostic evacuation.* This evacuation, a defini a meal, enables us to decide whether or not the stomach ha task of propelling the food at the right time into the intesti of motor activity, the *test-meal* is unquestionably more sati the test breakfast, for the reason that, in consequence of the put upon the stomach, we get a better idea of its activity. trial-breakfast may also be utilized to test the motor activity.

A healthy stomach is empty six to seven hours after th test-meal. If, after this time, remains of food are presc activity is impaired.

The greater the amount of food still present in the stoma the degree of motor insufficiency. We may, of course, diff ing degrees of motor insufficiency according to the amount nd the time it remains in the stomach. Usually it is sufficient to differentiate *two degrees,* in one of which the stomach is not yet empty seven hours after a test-meal but is so in the morning; in the second, a more advanced stage, in which, after a simple evening meal, the stomach is not empty on the following morning.

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[1] *Verhandlungen des XVIII. Congresses für innere Medicin,*

My usual method is this: In the evening, seven hours after the midday meal, I evacuate the stomach. If there are profuse remains of food motor insufficiency is proven. I then wash out the stomach and permit the patient to take a simple evening meal. The stomach is evacuated the next morning before any food is taken. If it is empty, this shows that the stomach which was unable to digest a test-meal in seven hours is, nevertheless, able to digest a smaller meal, the evening meal, in a longer period —in about twelve hours. If the stomach is not empty upon the succeeding morning, a higher grade of motor insufficiency is evident.

Another method is to wash the stomach clean in the evening, after which the patient is permitted to take the evening meal, and the stomach is evacuated the next morning before any food is taken. The next day, without a previous evening washing, the supper is eaten, and the following morning the stomach is again washed out before any food is taken. In the latter case we will often see, before food is taken, that the stomach still contains remains of food, while, upon the day before, when an evening washing preceded, it contained none. This shows that the evening washing has benefited the condition of the stomach. Naturally this method may be modified in various ways.

Higher grades of motor insufficiency wherein the stomach, even overnight, cannot completely get rid of its contents, as a rule are associated with an increase in size, and this is not surprising. Evidently, the duration of the motor insufficiency has a causative influence in the development of this increase.

In connection with gastric evacuation, there must be an exact examination of the gastric contents. This also occasionally aids us in the diagnosis of motor disturbance, for, very frequently, remains of ingesta which had been consumed days, even weeks, prior, are evacuated,—substances such as cherry-stones, seeds of cranberries, of cucumbers, of pears, currants, etc. Marked gas fermentation also presupposes a prolonged stagnation. The three characteristic layers of the gastric contents (the upper or foamy layer, the medium or fluid layer, the lower or finely distributed starchy sediment) proves stagnation and at least a sufficient HCl secretion. For an exact estimation of slight disturbance in less developed cases Strauss[1] gives valuable advice, which is to take the unfiltered gastric contents, place them in a fermentation tube with grape sugar, and permit them to stand for some time; by this means we get an idea of the rapidity and intensity of the gas formation, of the retained causes of fermentation. examination of the gastric contents does not aid so much estimation of motor insufficiency as in the question of the na cause of the dilatation, that is, of the underlying affection. this process only can we understand the nature of the tran the ingesta, and from the gastric juice only can draw conch ing the underlying affections which have caused the dilatati insufficiency, nevertheless we obtain from this, particularl in connection with the other symptoms, important point Thus coarse undigested remains of food, particularly of me free HCl and profuse amounts of lactic acid primarily indic while a profuse residue consisting only of fluid and rem with decided yeast fermentation, the presence of large am cells which are beginning to germinate, as well as sarcinæ an amount of hydrochloric acid, favor benign dilatation.

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[1] *Zeitschrift für praktische Aerzte*, 1896, Nr. 6.

A constant admixture of bile in the material vomited or e the stomach points to a deeper seat of the obstruction, to an duodenal stenosis.

In the majority of cases, we may content ourselves wi motor activity by simple lavage, and, perhaps, as a control lavage may be performed. If, one hour after a test breakf seven hours after a test-meal, a plentiful residue of half a is found, motor insufficiency is certainly present.

This method is satisfactory in practice. For exact scientific purposes it is insufficient. Here the method mu time, show us what residue remains in the stomach. Form mining the residue have been given by Mathieu and Rem Goldschmidt,[3] Sörensen and Brandenburg.[4] We cannot en tails of these processes. In practice they may all be dispens

Any one who desires to employ still other methods of purposes of control finds a number of such at his service. currant test, in which the patient is given in the evening of currants or cranberry jelly, and upon the succeeding stomach is evacuated after a test breakfast or a test-meal ination is made to determine whether, and how many, seeds in the evacuated contents. In a normal person these are n Other tests are by the *salol method* and Klemperer's *oil* tests are all inferior to the old process, first employed testing the duration of digestion. The *iodipin* method,

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[1] *Soc. de biolog.*, 1890.

[2] *Therapeutische Monatshefte*, 1895.

[3] *Münchener med. Wochenschrift*, 1897, Nr. 13.

[4] *Archiv f. Verdauungskrankheiten*, Bd. III.

which depends upon the fact that the gastric juice is incapable of freeing iodin from the iodin fat, and that the iodin is first set free in the intestine, can, if necessary, be employed as a substitution method, which is not quite free from objection in those cases in which absolutely no use can be made of the stomach-tube. This method has the disadvantage that it consumes more time than lavage, while the latter is superior in that it enables us to form conclusions in regard to the motor activity, and also shows us the secretory condition.

These are the *objective* signs which indicate gastric dilatation and motor insufficiency.

Before we ask what aids we have at command to assist us in determining the *nature* of the dilatation and the motor insufficiency, a few symptoms may be briefly mentioned which are directly connected with the disturbed motor activity of the stomach.

That disturbances of *intestinal activity* are not rarely observed in dilatation of the stomach is not surprising. Patients with this affection frequently have a tendency to constipation; much more rarely they suffer from diarrhea. This constipation may, to a great extent, be due to insufficient absorption of water; the excretion of water from the stomach and the difficult propulsion of the ingesta into the intestine may be the cause of this. In cases of high-graded dilatation the dryness of all the tissues is the reason why more water is withdrawn from the intestinal contents than in the norm. This readily explains the conspicuously hard and dry composition of the feces so often observed, and the difficulty of their propulsion.

The *diminution in the amount of urine* noted in extreme dilatation is due to the decreased absorption of water. The amount of urine voided is a fair measure of the activity of the stomach. In very marked cases, the daily excretion of urine is often decreased to a few cubic centimeters. An increase in the amount of urine is an infallible sign of improvement; the slighter the amount, the more concentrated it naturally is, and the higher its specific gravity. In dilatation with a plentiful HCl production, the urine often shows a neutral or alkaline reaction, and also when, in consequence of profuse vomiting or by frequently repeated lavage, much acid gastric juice is removed.

Occasionally we note an action upon the *heart* shown by a *slowing of the beat*. However, this occurs not only in dilatation, but also in many other gastric affections, and, therefore, may in nowise be designated a pathognomonic peculiarity of dilatation.

More important than the previously mentioned effects are certain derangements of the *nervous system*. Among these the symptom-complex designated as *tetany* is especially interesting. This, as is well known, manifests itself particularly by tonic muscular spasms in the hands and arms, occurring intermittently, and an increased irritability of the nerves.

Fortunately, tetany is not among the frequent sequels of dilatat cases of tetany in gastric dilatation observed up to the present ha in benign pyloric stenoses, mostly in such as were accompanied acidity and acid fermentation; however, cases of tetany have observed in carcinomatous pyloric stenosis, in absence of HCl sec in lactic acid fermentation. How tetany is produced in these c a mooted question. With Kussmaul, some attribute it to a de water in the organism; in another theory, the development of of spasm is attributed to a reflex irritation of the nerve cente irritated nerves of the gastric mucous membrane; in a third the tetany is regarded as the consequence of an intoxication of th by abnormal products of metabolism in the diseased gastrointes So far as I am able to judge, the last theory has found the mos A discussion of these various theories would lead us too far fro ject; and, naturally, I must decline to enter upon a special de the symptoms of tetany. (See volume " Diseases of the Nervou

Headache and vertigo are not rarely the sequels and ac symptoms of dilatation; usually these are not very intense. minal stages of marked dilatation the patients are sometimes become soporose, and finally succumb to coma. Such a case, in and general spasms occurred and terminated fatally after a fe recently been reported by Jürgensen.[1] In this case the fac worthy that during the soporose condition the secretion of entirely. The supposition of an autointoxication in such cases very reasonable.

The appearance of curious *drumstick-like changes in the toes* in a case of gastrectasis due to benign pyloric stenosis m tioned. This case, recently reported by Dennig,[2] is interes reason that after a successful operation (pyloric resection) complete retardation of the drumstick-like changes.

If, after an examination by the methods detailed, gast motor insufficiency have been determined, we then inquire w duced the condition, whether a mechanical obstruction at, or borhood of, the pylorus, or simple flaccidity. The question m be decided at a glance, or immediately upon palpating the other cases only after minute consideration of all the sympto prolonged observation. If the condition were as simple as been maintained, that every chronic dilatation is due to a passage or to a local process at the outlet of the stomach, the ectasis, and also of "pyloric stenosis" could be at once ma conditions are not so simple, although it is true that the grades of dilatation occur in pyloric stenosis. In practice, however, we must diagnosticate not only the marked grades of ectasis, but also the milder grades, the earlier stages.

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[1] *Deutsches Archiv f. klin. Medicin*, Bd. LX.

[2] *Münchener med. Wochenschrift*, 1901, Nr. 10.

Such forms are due to an atonic condition. But spastic processes also, without an organic stenosis being present, occasionally, as we have before stated, play a rôle in the etiology of dilatation.

Atony, as we have seen, occurs in persons whose nutrition has suffered, and in those with flaccid abdominal walls; not rarely, gastroptosis and enteroptosis exist simultaneously. Such an atonic stomach, as a rule, has a flaccid feeling with but little tension. In spite of a sufficient secretion of gastric juice, as shown by lavage, we frequently find coarse particles of food, especially fibers of meat. The latter are probably due to a loss of energy of the muscularis which prevents an intimate admixture of the ingesta with the gastric juice.

A successful treatment, in such cases, often confirms the diagnosis. If, by suitable diet and by improving the nutrition,—briefly, by methods which strengthen the tonus of the muscularis and improve the general nutrition,—we can remove the symptoms of motor insufficiency, this result favors the view that we are not dealing with an organic stenosis.

DIFFERENTIAL DIAGNOSIS

In the *differential diagnosis between atonic and mechanical dilatation*, the history is important. When symptoms of ulcer have previously existed, when in connection with this the picture of dilatation has gradually developed, our first thought is that a cicatrix of the pylorus has produced stenosis. If examination reveal a uniform resistance in the pyloric region, if the secretion of gastric juice be normal or increased, if we observe active peristaltic movements, a cicatrix from ulcer is indicated. When, upon inflation, we find that the thickened area remains immobile at the same place in which it appeared while the stomach was empty, this we assume to indicate a simultaneous perigastric adhesion.

The diagnosis is more difficult in cases in which *hypersecretion and dilatation* exist simultaneously. I have already discussed the varying possibilities of this combination. Active peristalsis favors a mechanical obstruction, although not necessarily an organic hindrance. Preceding gastric hemorrhage favors ulcer, and, possibly, a cicatricial stenosis, although this is by no means absolutely proven. The results of therapy often indicate that the cause which brought about the dilatation could not well have been an organic stenosis.

Probably the most positive sign of mechanical obstruction at the pylorus is *active peristalsis*. It is never absent except in the terminal stages and in those instances in which secondary degeneration of the hypertrophic musculature has occurred. Yet the obstruction is not necessarily an organic one. As is well known, spasms of the pylorus play a part ir of disease of the stomach as well as in dilatation, and are parti inent in those which run their course with increased secreti juice. According to Serejukow, acids cause the sphinct contract.

A factor not without significance in the differential diag these forms of dilatation is the *rapidity of the influx and* gastric contents; that is, of the water used for lavage. Th tonus of the stomach, the more powerful the musculature in at the outlet of the stomach, the greater the force with wh will empty itself. The more slowly the water used for la the more rapidly will it flow out. The conditions are the with very flaccid, thin, atrophic gastric walls. Here the g are discharged very slowly and incompletely; the water poure rushes with force through the tube, so that swirls develop, an aspirated into the stomach.

An admixture of *bile* is not infrequently observed at t of a washing of the stomach, especially in atonic dilatation. of stenosis exist, the permanent regurgitation of bile favors lary seat of the stricture.

The most important sign of stenosis, however, is always *tion of a tumor* at, or in the vicinity of, the pylorus. The t does not prove a stenosis, for we must be certain that it stomach or to the adjacent portion of the duodenum.

When a tumor has been diagnosticated, the question to is whether it be benign or malignant. We would digress entered upon this differential diagnosis. This point can onl by considering the duration of the disease, the condition contents, the secretion of the gastric juice and the like.

That the *results of treatment* may be utilized to a ce diagnosis, particularly in differential diagnosis, whether ste or only functional spasm be present, is clear. Rapid relie tion by suitable treatment favors the atonic form or Naturally, in a temporary improvement, we should not at c selves to make this diagnosis. Ectases due to organic s ameliorated by methodical lavage, by diet, and other ren improvement is not permanent.

COURSE OF THE DISEASE

Having described the symptoms of motor insufficiency it is next expedient to describe their course. In the majo clinical picture of dilatation develops gradually; rarely do a single deleterious effect, brief in duration, suddenly c

In the course, as in the development, we must differentiate between *acute* and *chronic dilatation*. The acute varieties, as a rule, are rapidly cured by appropriate treatment; yet, repeatedly, cases with a fatal termination have been observed. In explanation of these, Albrecht's previously mentioned investigation of arterio-mesenteric intestinal occlusion at the duodeno-jejunal boundary might well be considered.

Whether, in all cases of acute gastric dilatation running a fatal course, mechanical factors of this kind play an important rôle, cannot, naturally, be subsequently decided. The symptoms of severe acute dilatation have several times appeared without any demonstrable cause. In a few cases the clinical picture developed directly after anesthesia; in several cases a severe infectious disease immediately preceded the condition. It is worthy of note that in a number of these cases vomiting was absent during the entire course; this is probably to be explained by the high-graded, over-distention and loss of tonicity of the walls of the stomach.

Such severe, rapidly fatal cases are very rare. Much more frequently mild and moderately severe forms are observed, the origin of which is traced to gross errors in diet. A marked, painful swelling of the abdomen soon occurs and is associated with nausea, vomiting, and great thirst. In mild cases the symptoms disappear in a few days, particularly if thorough lavage is performed, and we must be careful to see that the stomach is absolutely emptied. In other cases the symptoms do not yield so readily. The earlier and more energetic the treatment, the more favorable and rapid, as a rule, is the course.

In private practice, we more frequently deal with *chronic* cases—dilatation which develops gradually. The course of these varies greatly, and depends upon different factors: the underlying cause, the mode of life, and numerous other conditions. Not infrequently we notice a change in the symptoms, sometimes amelioration, at other times aggravation; this is observed, above all, in cases of non-organic stenosis. Organic stenosis, on the other hand, generally shows a progressive increase in the symptoms. The most marked degrees of dilatation are met with in pyloric stenosis; atonic dilatation, in the majority of cases, is extremely slight. Yet it appears to me that we are not justified in denying absolutely the occurrence of atonic dilatation, as some writers do. At all events, it is rare, and usually not of marked extent.

It is unfortunate that the test by physicians of the motor activity has not everywhere been regarded as of equal value with the test of the secretory activity. Nevertheless, we must admit that the disturbances of motor activity are often of the greatest importance, even more so than the variations in the secretion of the gastric juice. If in every serious gastric disturbance the same importance were attached to the motor as to the secretory activity, many a case which is now recorded under the name "chronic catarrh" or "nervous dyspepsia" would be recognized as motor insufficiency or as dilatation, although not of extreme degre met with a considerable number of such moderate dilatations w appropriate treatment, proper diet, rest, forced feeding, metho and the like, were entirely cured. These patients with atony, relatively light meals, have a sensation of satiety, of pressure in the stomach; they feel indisposed to follow their occupa are listless. As a rule there is no complaint of actual pai troptosis goes hand in hand with atony, as it frequently do turbances are increased. The patients, after eating only a littl very disagreeable sensations, they lose their appetite, and gr nutrition suffers. Such atonies are very often disregarded, or diagnosticated, and if they do not early come under suitable tre rapidly increase in severity. Only a minute objective exam the aid of an accurate history make an early diagnosis possible. promptly suitable treatment is instituted, the easier is the c be readily understood that these forms show frequent changes, to relapse, and that periods of amelioration and aggravation al

A much more uniform course is generally observed in motor and dilatation due to organic stenosis. Here, also, transito ment may occur; but it cannot last long unless we succee ing the obstruction which causes the stenosis; however, this is ble except by surgical interference. We cannot enter into d modifications of these forms on account of the great variety of

DIAGNOSIS

In discussing the diagnosis of motor insufficiency in dilat be brief. This is based solely upon the *objective* examinati *proof of prolonged digestion, and upon an increase in the stomach.* Among the methods that have been mentioned, the one for determining motor insufficiency is to wash out the to seven hours after a test-meal, or two hours after a test breakf ing sounds heard at a period in which the stomach should be the diagnosis, but never render the diagnostic washing out The latter has the advantage that it simultaneously enable conclusions in regard to the secretory activity, and also to de processes cause the dilatation.

The *degree of motor insufficiency* is readily estimated fro of retained material and the period of its retention in the st

Considering the diagnostic criteria which have been me fusion with other affections is hardly possible. *Megalogastr* be mistaken for dilatation if we should neglect the washi stomach which is absolutely necessary to test the motor a less would it be possible to confound the condition with ch or with nervous dyspepsia. This confusion is impossible if diagnostic lavage be performed. However, atony and motor insufficiency may occur simultaneously with chronic gastritis and nervous dyspepsia; motor insufficiency is only a disturbance of function which may develop in various ways.

In practice confusion of gastrectasis with *gastroptosis* occurs. This error is not rare, for the reason that with a low position of the greater curvature of the stomach and a succussion sound developed below the navel dilatation is assumed without further investigation. This is not justified by the facts. The proof of displacement of the lower boundary of the stomach is not sufficient to determine dilatation; its total extension must be determined, not only that of the lower, but also of the upper and the lateral borders. The investigation of the latter is interesting. As Michaelis [1] has shown in cases of enlargement of the stomach in which the gastric motility was severely disturbed, the right border, as a rule, is found much farther removed from the median line than in a stomach with normal motility.

On the other hand, we must remember that motor insufficiency and dilatation are often combined with gastroptosis. It is quite natural that a permanent over-weighting of the stomach should lead to a sinking, to gastroptosis. In fact, in almost all extreme dilatations, we find more or less sinking of the lesser curvature. An extreme case of decidedly low position of the stomach which may readily be recognized (with a high grade of dilatation) may be seen in the accompanying illustration (Fig. 10) in the case of a woman, aged 59, who had carcinomatous stenosis of the pylorus.

Simple atonic dilatations very frequently are combined with gastroptosis. This combination is not difficult to recognize, provided we make use of the previously mentioned diagnostic aids.

By what means atonic dilatation is to be distinguished from that caused by mechanical obstruction has been stated. This question can often only be decided after prolonged observation, under some circumstances only by studying the results of treatment.

Much more difficult is it to decide as to the nature of the constriction. Whether we are dealing with *benign* or *malignant* stenosis is a most important question, and, in the majority of cases, not difficult to decide. A hard, nodulated tumor in the pyloric region, abundant coarse residue with frequent hemorrhagic admixture in the form of coffee-ground masses, absence of free HCl, lactic acid fermentation, and rapidly increasing cachexia, naturally favor malignant stenosis. But, in malignant stenosis, particularly in such as develops from an ulcer, we often find a normal secretion of gastric juice. It must be borne in mind that *no single symptom is decisive*, but the combination of all the symptoms is necessary to a diag-

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[1] *Zeitschr. f. klin. Med.* XXXIV.

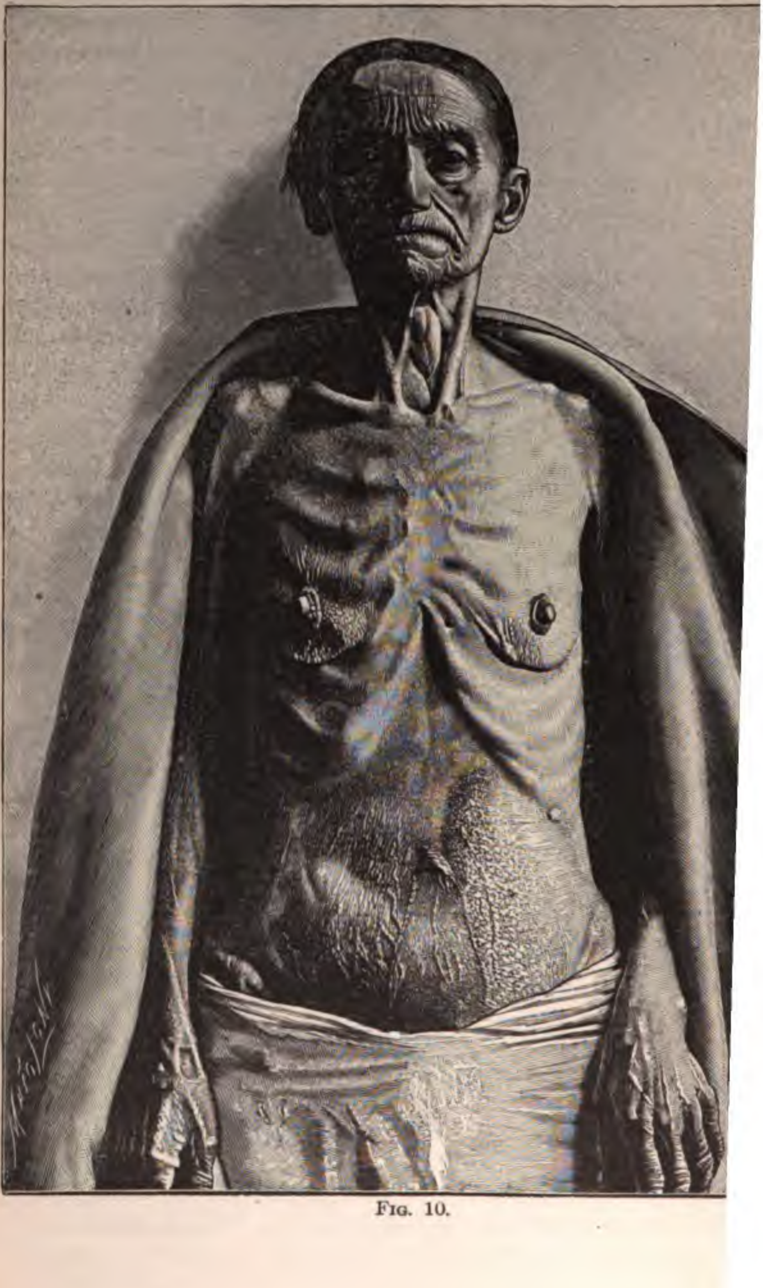

FIG. 10.

nosis; whether one or the other is absent is not conclusive. But a diagnosis can never be looked upon as certain which does not combine all the symptoms.

PROGNOSIS

As has been shown, the importance of motor insufficiency and dilatation varies greatly in the individual case; it may run an *acute* or a *chronic* course. At one time it represents a transitory atonic condition, at another time it depends upon a mechanical obstruction, which in one case may be benign, and in the other malignant. The prognosis of motor insufficiency in dilatation, therefore, also varies strikingly in the individual case. Some forms are cured in the briefest time by proper management, others only after months or even years of treatment; some in the briefest time terminate fatally, others again are not susceptible to internal treatment, and surgical measures alone will cure the affection or moderate its consequences.

The earlier treatment is begun, and the more systematically it is carried out, the more rapidly do *acute* dilatations improve. Under some circumstances threatening symptoms may intervene, and the cases even terminate fatally. Generally no prognosis is possible in *chronic* dilatation. Many factors influence the prognosis in a given case; thus, the duration of the affection and the degree of motor insufficiency. Moderate degrees of motor insufficiency which have not existed for a long time naturally lead to a more favorable prognosis than long-existing, high-graded dilatations. But, above all, the prognosis depends upon the underlying *cause* of the dilatation. If we succeed in removing the causative factor, as a rule we also succeed in ameliorating the dilatation. Mild and medium grades of atony under suitable treatment are often cured in the briefest time. Where the cause of the dilatation is an organic obstruction in the pylorus or its vicinity, improvement can only be brought about by surgical means. Internal treatment in such cases produces apparent, but in reality transitory, results. Unfortunately many cases are no longer suitable for operative interference, such as extensive carcinomata, or those in which metastases are present and the like. The dilatation alone does not permit a positive prognosis, but this depends, above all, upon the underlying affection; that is, upon the cause which has produced the dilatation.

TREATMENT

We now come to the main question: *How shall we treat dilatation?* Naturally it is not my object to explain here all the methods of treatment and the other procedures which may be necessary on account of the causative factor, carcinoma, cicatrix from ulcer, perigastric adhesions, and similar conditions; we should digress too far from our actual theme. But we shall consider only the treatment of motor insufficiency anc as such.

Acute dilatations which have been brought about by over- stomach with food difficult to digest require immediate and thoro ing of the stomach and an absolute rest of the organ for some ti debility necessitates the administration of food or fluid, rectal a should be resorted to. Under all circumstances, however, t must have absolute rest in the days succeeding. How long thi is to be pursued naturally depends upon the severity of th complex. In acute dilatation in which symptoms of obstruct it is not enough merely to empty the over-filled stomach, and for some time the administration of food and fluid by the mou also attempt to relieve the coils of the small intestines at thei roots, which, by a drawing process, have been forced down int For this purpose, the patient must be placed in such a posit small intestine may find its way out of the pelvis, and the te mesentery be quickly relieved. Therefore the knee-elbow posit prone upon the stomach, is most effective. Cases of this kin recovery have recently been reported by Müller.[1] Where this not bring relief, eventually surgical measures only can avail.

The conditions are different in the much more frequent c of dilatation and motor insufficiency with which the physician concern himself. Although our object varies according to wh dealing with simple atony, with atonic dilatation, or with dila by a mechanical obstruction, nevertheless, certain therapeu equally operative in all these forms. In these groups, prolo tion, and all abnormal weighting of the stomach, must be avo as possible, that is, combated, and the diet must be so ordere necessitate the least possible exercise of the motor activity of and can by no possibility be injurious.

Our primary object in dilatation is to regulate the diet. variably consider first, the kind of food, and, secondly, much tant in dilatation, the form in which it is administered. In tice the majority of physicians advise a dry diet in dilatati to be refrained from as much as possible, for they load th their weight, are badly absorbed, and, in pyloric stenosis are the stomach with difficulty as is proven by the over-distention c with fluid. A dry diet, therefore, with the greatest possible fluids, is advisable.

In every form of dilatation, whether it depend upon a mechanical obstruction, the motor activity of the stomach either absolutely or relatively insufficient, according to the ar required of it. We must reckon with this motor weakness; i. e., we must choose a diet which necessitates but slight exertion on the part of the motor power, which does not require over-exertion, but one that prevents it. For, by means of the motor power, the fragments of ingesta are split up in the stomach, reduced in size, liquefied, and, finally, propelled from the stomach into the intestines.

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[1] *Deutsche Zeitschrift für Chirurgie,* Bd. LVI.

v. Mering, by his well known researches, has proven that the healthy stomach possesses only very slight power of absorption, and that its labor consists chiefly in propelling the fluid gastric contents into the intestines. Moritz, too, has demonstrated by investigations that fluid food is more rapidly propelled into the intestine than pappy food, and this again in a shorter time than solids.

Therefore, above all, in motor insufficiency and dilatation we must choose a diet which is readily forced onward into the intestine. This is true, however, only of fluid and pappy food.

In opposition to the almost universal practice of employing a dry diet, I have for years advised *fluid* and *pappy forms of food in dilatation.* This method appears to have recently gained many adherents. In the choice of the diet we must always reckon with the weakened motor power, and, therefore, so far as possible, choose a food that may be broken up into fine particles.

One objection has been raised to fluid diet, namely, that the nutritive value of fluid food, in proportion to its volume, is but slight. This argument may, however, be met by choosing a food as nutritious as possible. Such a nutriment is represented by *milk,* which may be given pure, or as buttermilk, as cream, as milk-custard, and as gruel with various additions of grits, rice, and the like. The taste may be varied by the addition of aromatic substances—by brandy, lime-water, by the addition of cocoa, vanilla, cinnamon, lemon, etc. We can generally succeed, in almost all patients, in administering larger or smaller amounts of milk, even when they express a dislike for it.

Eggs may be variously prepared and, in addition to other foods, take a place in the diet of the patient. The addition of tropon, nutrose, plasmon and similar preparations to fluid and pappy food increases their nutritive value.

Meat, in so far as the underlying affection permits its use, had best be given scraped or chopped. Calves' brains and sweetbreads, on account of their pulpy consistence, are very suitable for such patients. Game must be avoided, as well as goose, duck, and fat pork. Of fish the most suitable are giant pike, pike, perch, and shell fish. Vegetables are to be given only very finely split up or in the form of purée,—of asparagus the heads, of cauliflower the tops, and mashed potatoes.

Fat, especially milk-fat, is also suitable for patients with disturbances of the gastric motility, particularly for such forms as are combined with hyperacidity. Formerly the opinion prevailed that large amounts of fat lie heavy in the stomach[1] and that in disturbances of its motility they should be avoided. The researches of Strauss,[2] Bachmann,[3] Wolkowitsch[4] and others, show that too much cream will diminish the secretory power of the stomach, and even in the stomach showing motor insufficiency it is subjectively better borne than a large quantity of carbohydrates. In hypersecretion, and particularly when disturbance of motility is simultaneously present, the profuse ingestion of undissolved carbohydrates increases hypersecretion. For these cases of disturbance of motility with well maintained HCl secretion, it, therefore, seems more rational to substitute for the carbohydrates as far as possible the fats which are readily and thoroughly broken up. Here the *albumin fat diet* for several weeks is advisable.

It is well known that some patients cannot digest fat, particularly cream. In these cases of motor insufficiency and dilatation, the opinion previously maintained that fat must be prohibited and that, at most, but small quantities of butter should be permitted, is no longer in accordance with our clinical experience.

A definite *diet list* for patients with dilatation cannot be given; it is clear that wealthy patients may have a more varied dietary than the poor, but for both the rules given above, which primarily enjoin a rest for the stomach, are alike operative.

Another self-evident rule is that such patients should *never take too much food at one time.* The custom of eating only two meals a day is not suitable for patients with gastrectasis. The meals are to be small, but they should be frequent. Under some circumstances, the method of Albu[5] of giving the patient fluid, concentrated food through the stomach-tube immediately after the morning and evening stomach washing may be adopted; but this would come into consideration only in the nutrition of very debilitated patients with marked anorexia.

In very emaciated persons, to improve the nutrition and to rest the stomach as much as possible, a portion of the fluid food may be given by the rectum. If we are only endeavoring to combat the decrease of water in the organism, enemata of water with the addition of salt (1 teaspoonful of table salt to a liter of water) or a small quantity of brandy, or wine bouillon enemata ($\frac{2}{3}$ bouillon and $\frac{1}{3}$ Rhine wine) may be given. It is advisable to give nutritive substances admixed with the enema. For this rpose a peptone-enema (50 to 60 of peptone to 300 of milk), milk and g enemata (3 eggs with the addition of 3 grams of table salt, in 300 of ilk) and starch enemata (50 starch to 300 of milk) may be employed. olutions of sugar are liable to produce irritation of the mucous memrane of the rectum. If they must be employed, not more than 15 or 20 rams of sugar to 300 of fluid should be used. Nutritive enemata of this ind must of course only be given in extreme dilatation, and for a short ime; as complete substitutes for the administration of food by the mouth, hey naturally fail. Subcutaneous injections of fat may, under some circumstances, be given for nutritive purposes.

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[1] In regard to the question of the part played by the stomach in the digestion of fats, I refer to the investigations of Volhard upon "Absorption and Fat Splitting in the Stomach." recently published from my Clinic. (*Münchener med. Wochenschrift*, 1900, Nr. 5 u. 6. und *Zeitschr. f. klin. Med.*, XLII.)

[2] *Zeitschrift für diätetische und physikalische Therapie*, III.

[3] *Archiv für Verdauungskrankheiten*, V.

[4] *Wratsch*, 1898, Nr. 13, in *Archiv für Verdauungskrankheiten.*

[5] *Deutsche med. Wochenschr.*, 1900, Nr. 11.

Alcohol is generally to be avoided in dilatation. It is true the stomach will absorb alcohol, but, with its absorption, a plentiful excretion of water from the stomach occurs, which is the greater the larger the amount of alcohol absorbed.

Although these diet rules apply in general to every form of dilatation, whether it run its course with normal or defective secretion of gastric juice, nevertheless, this secretion of gastric juice should be considered in the choice of food. Where it is more or less decreased, milk foods, strong soups, and the most tender meats are indicated, the meat to be finely chopped or divided. But in hypersecretion and hyperacidity, the carbohydrates must be limited, and a meat-fat diet is preferable. In cases of this kind where the secretion of gastric juice is due to pyloric stenosis, where, therefore, notwithstanding dilatation, gastric peristalsis is still active, it may be expected that the stomach is still able to digest meat divided less finely. At all events we need not be so careful in this respect as in the cases with diminished secretory power and simultaneous atony.

As with every damaged or weakened organ, so in the case of the stomach it must be our first object to give it rest. The diet should be light, but, nevertheless, sufficient food must be taken. Rest of the organ is facilitated if the patient with dilatation is kept as much as possible in a recumbent posture.

That an upright position of the body is more unfavorable for the dilated stomach than a recumbent one is clear. After eating, the patient should lie down for a long time. Lying upon the right side after eating has frequently been recommended to facilitate the propulsion of the food mass into the duodenum. It is self-evident that such patients must *eat slowly and masticate thoroughly.*

If the dilatation and the motor insufficiency are due to the difficulty of expelling the gastric contents, we must resort to measures that will assist in this expulsion.

For this purpose systematic massage has been advised. Massage of the stomach, however, also serves the further purpose of strengthening the astric musculature. If we intend the massage merely to produce a more apid propulsion of the ingesta into the intestine, it should not be given immediately after a meal but a considerable time later. Mass traindicated in cases of decided fermentation, which must f moved. In the majority of cases the results of massage ar satisfactory. We cannot describe the technic. Massage of th however, should always be given by the physician.

In atony the *faradic current* is useful to strengthen the of the stomach; this, as a rule, is employed extraventricularly, intraventricularly. Certain *hydropathic procedures*, douches, douche, and, particularly, the so-called Scotch douche, are From my experience I cannot advise internal douches; in ext tion I do not consider them free from danger, and in milder simple methods may be substituted.

To promote more rapid propulsion of the gastric conten intestine in cases of organic or spastic stenosis of the pylorus, olive oil has recently been recommended. Cohnheim[1] advise one hour before meals three times daily. Not only in case of the pylorus, but also in cicatricial stenoses of the same, h have had good results. In several of these cases, there wa hope of recovery except by surgical interference.

All of these methods are designed *to rest and strengthen* t they may suffice in mild grades of motor insufficiency and not in the severe forms of dilatation in which the stomach i fectly empty, but even in the morning, before taking food, por consumed the previous day are still present. Here it is nec lieve the stomach of its abnormal contents by *gastric lavage*. forms of motor insufficiency and dilatation, *gastric lavage* is valuable and indispensable aid.

The degree of motor insufficiency which necessitates met is a disputed point. All authors are unanimous that, in forms in which the stomach in the morning before breakfast from the day previous, it should be washed daily. It is slighter dilatation. In my opinion, even mild forms of dilata regular lavage. The normal stomach acts only periodically, ously. Six to seven hours after the midday meal it is empty, five hours after a simple evening meal it should no longer In consequence of this, the stomach has certain periods of re eased stomach with motor insufficiency should have no great no longer hours of labor, than correspond to the norm. If in the evening, before supper, still contains larger or small of food, it must be washed out. If this is not done it is abno and if the debilitated and distended stomach with motor i not to be still further stretched and taxed, naturally no mo be exacted from it than from a normal organ. This would, however, be the case, if we expect an evening meal to be digested besides the remains of the midday meal which is still in part undergoing fermentation. This would not ensure to the diseased organ the rest which is of such primary importance.

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[1] *Archiv für Verdauungskrankheiten*, Bd. V.

If the stomach that has been washed before supper is empty upon the following morning, this *evening lavage alone* will be sufficient; if not, it must be performed in the morning in addition. This is not only necessary to relieve the stomach, but because abnormal fermentation and decomposition occur upon prolonged stagnation of the ingesta.

Many recommend lavage in the morning before breakfast. They believe that if the stomach is not empty at this time it should be washed out, and that this is the best time for the procedure. They also believe that at this hour the washing can be most rapidly performed, and is the least exhausting. I do not coincide with this reasoning. Lavage should relieve the stomach by removing its decomposed products of abnormal fermentation.

It is undeniably true that lavage is most easily performed in the morning, and more rapidly because the stomach before breakfast contains relatively the slightest residue of food. But this method gives little relief to the stomach; it has no periods of rest as in the norm. It is better, as others advise, to carry out the lavage late in the evening between 9 and 10 o'clock. This gives absolute relief and an actual period of rest follows. To me it appears most rational to use lavage immediately before the evening meal. If, after seven hours, much residue is still present, which must, under any circumstances, have begun to ferment, it is wiser to remove this and to introduce fresh food into the cleansed stomach than, as the exponents of lavage in the late evening recommend, to add an evening meal to the fermenting remains and evacuate the stomach two or three hours later. If, after evening lavage, the stomach is not empty upon the following morning, it must, in addition, be washed in the morning.

Therapeutic washings should be as thorough as possible, not only to empty the stomach, but to remove as well the causes of fermentation. This should be done first with lukewarm water; medications may be necessary in subsequent washings on account of special individual conditions, because of acid, abnormal fermentation, and the like. The latter, especially, often renders the use of drugs necessary, such as salicylic acid (1–1000), boric acid, resorcin, and similar antiferments.

A thorough cleansing of the stomach is more rapid when there is relatively increased gastric tonus, such as we meet with in pyloric stenosis rather than in the atonic forms. Naturally, lavage must be continued until the stomach is thoroughly cleansed, or until we have convinced ourselves of the uselessness of further effort.

To the ***physical aids*** which play but an indirect rôle i of dilatation, *bandages* may be added. They do not redu tion, but they give the dilated stomach a firm support; t assist in producing subjective amelioration, but to a certai relieve the condition. They are particularly valuable in th cases in which, besides dilatation, there is also gastropto there is, at the same time, a marked degree of flaccidity of walls.

These are the chief remedies and methods to which we tation of the stomach. Actual *drug medication* for dilata come into question. It has been maintained of some rem *strychnin*, that they stimulate the tonus and peristalsis of which has also been claimed for *creosote, orexin* and other I have observed no remarkable action of this kind from any o applied.

A few words must be devoted to the *belladonna pre* to *atropin* since these remedies have, to a high degree, an effect. Spasm of the pylorus, however, often exerts an infl tation, particularly in those varieties running their cours acidity and hypersecretion. As I have shown, the bellad tions not alone inhibit the secretion of gastric juice, but ar time, antispasmodic. On account of these properties, the ployed with advantage in the two forms of gastrectasis eve as symptomatic remedies.

The *alkalies* also, even if only indirectly, are useful in c dilatation, especially in those varieties in which there is Here they prevent the abnormal production of acids, and digestion of starches as well as the propulsion of the gastri

I cannot enter minutely into an enumeration of drugs of *individual symptoms*; they do not affect the dilatatio only certain symptoms caused by the underlying condition.

Tetany, a symptom-complex closely related to ectasis, discussed. Views regarding the origin of tetany and diverse. The majority of authors adhere to the intoxicati regard tetany as the consequence of an intoxication of the abnormal products of metabolism from the diseased gastro If this theory be correct, the therapeutic indication is that nation must be prevented. The active treatment of stagn ingly, the prophylaxis of tetany. Remedies which rel tetany, or prevent its occurrence, are unknown.

Unfortunately, the number of cases of dilatation in dies just mentioned do not bring relief is very great, a only hope of improvement or cure is in *surgical measu* serve two purposes, either in removing the actual cause o or in some manner bringing about the compensation of the damaged motor power.

By far the most frequent indication for surgical interference is seen in *pyloric stenosis*, immaterial whether the stenosis be due to a cicatricial narrowing, to a carcinoma, to compression from without or elsewhere. Where it is possible entirely to remove the stenotic obstruction, this should always be the primary aim.

When the surgical operation is decided upon, the proper time for it should be determined by a full consideration of all the conditions. Malignant stenosis should be operated upon as early as possible. Radical removal of a carcinoma is most desirable. Benign stenosis also necessitates surgical interference, but here we must usually wait until the uselessness of internal remedies has been proven. Where the internal clinician cannot ameliorate the condition with the remedies at his command, where, in spite of all the described methods it is impossible to introduce sufficient nourishment into the organism, the surgeon's art must be called into play.

If the radical removal of the stenosed area be impossible, prevention of the stenosis and compensation may be brought about by the formation of new outlets. *Gastroenterostomy* answers this purpose.

There are, however, also *relative indications* which are the result of special circumstances. Thus, we occasionally see patients whose condition is endurable so long as they remain in a hospital where proper diet and other auxiliary remedies are at hand to maintain them, and where they are able to help themselves. If they return to their former unfavorable surroundings, are compelled to work hard, and are unable to spare themselves, aggravation at once occurs. Here, the unfavorable circumstances of life, not the severity of the disease, compel operative interference which, under other circumstances, might have been postponed. Without an operation such patients as these would succumb in a very brief time. Here the operation is not absolutely indicated, but relatively, on account of the special environment of the case in question.

What operation (resection, pyloroplasty, gastroenterostomy, digital divulsion of the pylorus, etc.) is to be performed in the given case, can only be determined after opening the abdominal cavity. Radical removal of the obstruction which causes stenosis is primarily the aim, even though this very frequently cannot be attained.

Atonic dilatation also may, under some circumstances, although much more rarely, require surgical relief; this does not affect the atony, as such, but serves to facilitate the outflow.

Gastroplication cannot restore the tonus, but only decreases the volume of the organ, and in atony with this alone very little is attained. On the other hand, gastroenterostomy, while it does not remove the atony, prevents stasis, and under some circumstances restores the tonus of the organ. But, with the early recognition and proper treatment of atony, as a rule it rarely attains such a high degree of development tha intervention becomes necessary.

It is not our object to enumerate the individual surgical o the internal clinician must perceive the indications for surgery; of the method must be left to the surgeon.

GASTRIC ULCER AND GASTRIC HEMORRHAGE

By C. A. EWALD, Berlin

ETIOLOGY

In no other form of gastric disease is our insight into the finer pathologic processes and the methods which must be adopted for their cure so clear as in *gastric ulcer, round ulcer of the stomach,* or *peptic ulcer,* as the affection has been designated according to its peculiar features.

My actual experience regarding this disease cannot be questioned when I state that, in the last twelve years, I have seen and treated 1250 cases either in the Hospital, the Clinic, or in my private Clinic (not including my office practice). At this point, however, I do not intend to give a text-book description, but I shall consider chiefly the clinical aspect of the pathologic picture, and shall limit myself to the experiences of the last few years.

We differentiate the *acute* and *chronic* forms of gastric ulcer, not, however, by the different nature of the process, but by the rapid or slower course, and under this the various symptoms will be included.

Since Cruveilhier first gave us such a minute and comprehensive description of gastric ulcer (which had formerly been looked upon as a post mortem curiosity) that it constitutes a well-defined and easily recognized pathologic picture, clinical observers and experimental investigators have vied with each other in eagerly studying this affection.

However, the various experiments made in the course of time to discover the etiology of gastric ulcer cannot all be enumerated here. It must suffice to call attention to the fact that in the development of ulcer we are dealing either with the consequences of a mechanical, a chemical, or a thermic lesion of the mucous membrane, with a disturbance of the circulation at the point of lesion, or else such disturbances—and this is more frequently the case—have arisen without a preceding coarse lesion in a circumscribed, vascular area. A limited destruction of the tissue follows because the gastric juice attacks those areas which are no longer normally nourished, just as, to a fuller extent, it consumes the stomach after death, and under favorable conditions it produces softening. There is always, however, a pathologic disproportion between the composition of the gastric juice and the nutrition of the gastric mucous membrane, and the dogs, and, by severing the medulla oblongata, circumscribed hemorrhage into the stomach without the contractile power of the muscularis having suffered; besides, numerous cases are observed whose entire behavior excludes from the onset a flaccid condition of the stomach, and in which there was neither chlorosis nor anemia.

The *relation of the nervous system* to the development of gastric ulcer has been repeatedly studied. Lately Talma and his pupil, van Yzeren, have attempted to prove by clinical and experimental research that a spasm of the pylorus due to nervous influence is an etiologic factor. I have remarked elsewhere that this view, for several reasons, is most improbable; above all, because in the overwhelming majority of ulcers there is not the slightest indication of spasm. In my opinion van Yzeren confounds cause and effect, for he bases the results of vagotomia subdiaphragmatica in rabbits upon conclusions arrived at in man. Spasm of the pylorus, wherever present, is not the cause but the consequence of an ulcer. We have long known that conditions induced by hyperacidity, even where they run their course without producing ulcer, may lead to occasional or permanent spasm of the pylorus. Dalla Vedova has succeeded —as well as other authors before him—in producing necrotic, hemorrhagic, and ulcerative changes in the wall of the stomach of the dog by injuring the celiac plexus or its thoracic roots (splanchnic), the characteristic necrobiotic lesion closely resembling the peptic ulcer of man. From the nature and sequence of the injuries caused—hemorrhages of the gastric mucous membrane from the size of a pin's head to that of a lentil, hemorrhagic erosions, true ulcers and typical cicatrization—it is obvious that ecchymosis may be regarded as the beginning of the process from which other conditions arise.

The *development of gastric ulcer from trauma* has lately become especially interesting to us because the laws in regard to accidents frequently place us in a position where we must give an opinion as to the causal relation between trauma and ulcer. Many more or less trustworthy observations are distributed throughout literature which, in their totality, permit no doubt that gastric ulcer may, indeed, be produced directly by a blow, a contusion, a fall, or the like, which affects the gastric region. A comprehensive study of this aspect of the disease will be found in Stern's book, "Ueber traumatische Entstehung innerer Krankheiten," Jena, 1900.

I have repeatedly been called upon for such an opinion, and the following considerations have been my guide. First, we must ascertai whether immediately, or, at least, a short time after the trauma, a undoubted gastric hemorrhage occurred, for this is the only positive sig of a severe injury to the gastric mucous membrane. Directly followin this, nausea, anemia, syncope and localized gastric pain may occur; bu since, as is well known, such injuries of the mucous membrane he a blackish-brown extravasation of blood appears simultaneously with a loosening of the mucous membrane. Their number varies greatly. Occasionally they are so numerous in the region of the pylorus that the mucous membrane appears to be covered with them. From this erosion the typically chronic ulcer develops. But certainly not from those erosions which, as Langerhans emphasizes, in my opinion most justly, by their irregular distribution upon the mucous membrane, their numbers, and their confluence, differ widely from the typical ulcer with its characteristic appearance and its solitary seat. Langerhans states, "These hemorrhagic erosions usually develop from spasmodic contractions and coincident inflammatory processes; the round gastric ulcer, after a primary affection of an arterial vessel. Only exceptionally do erosions develop into chronic ulcers." But this author, and D. Gerhardt as well, accurately describe typical examples of the transition of such erosions into classical ulcers, and, as Gerhardt has shown, the small ulcers which develop from the swelling and rupture of lymphatic follicles may here be included.

According to the descriptions of Nauwerk, and the published cases of Letulle, Dieulafoy, Stokes and Krafft, the influence of bacteria, that is, of bacterial invasion, in the development of gastric ulcer, appears, on the contrary, to be of mechanical rather than of a true toxic nature, and we can hardly doubt that the lesions were originally bacterial emboli of the smallest vessels, upon the base of which the ulcer later developed, and this was the primary cause of a limited disturbance of nutrition of the mucous membrane. In the upper layers of the necrosed mucous membrane, various microörganisms collect.

A. Schmidt claims to have discovered an equally important factor for the development as well as for the cure of the ulcer in the possibility of contraction of the muscle situated below the lesion in the mucous membrane. Griffini and Vassale had already demonstrated that the healing of erosions of the gastric mucous membrane in animals takes place by the complete approximation of the edges of the wound, and thus further digestion by the gastric juice is prevented. Schmidt believes that the absence of such contraction accounts for the production of an ulcer from superficial losses of epithelium, because in experimenting upon a dog he was able to produce deep losses of substance though not chronic progressive ulcers whenever by suitable measures he prevented the muscularis in the area of the lesion from contracting. The conditions are similar in the human being, and the well known fact that gastric ulcer is so frequent in chlorosis and anemia might be attributed to the flaccidity (atony) of the gastric muscles. Admitting the latter to be true—chlorosis, in fact, promotes relaxation also in other organs, the heart, the intestine, the uterus—Schmidt's view has been by no means generally accepted. Years ago, I produced the same deep losses of substance in show a few uneven points, occasionally it is covered with bloody coagula or a greenish or brownish tough mucus. The size varies greatly, generally from that of a dime to a quarter of a dollar, but it may be very much larger, and sometimes may entirely cover the stomach. Its seat is preferably at the pylorus and on the greater curvature, which are the lowest portions of the stomach when in the erect posture, and those in which the gastric juice collects, so that Nolte has given the following scale of frequency: At the greater curvature 22, at the pylorus 13, upon the anterior wall 3, upon the posterior wall 2, at the cardia 1.

According to Brinton 42 per cent. of the cases are upon the posterior wall, 26.8 per cent. at the lesser curvature, and 15 per cent. at the pylorus. These reports are based on 220 cases. But such statistics are constantly modified by circumstances. In my experience, for example, ulcers of the pylorus are more numerous than those of the lesser curvature, and these are more frequent than those of the greater curvature. Gluczinsky coincides, for in 95 autopsies he found 63 cases of ulcer in the pyloric region. In the majority of cases there is but one ulcer; several, that is, three or more, are rare. Lange, however, in one case saw so many "that he was obliged to discontinue counting them."

Occasionally several originally distinct ulcers coalesce and form one large one.

From *microscopic sections* through the margin of a fresh ulcer the glandular tubules are seen trough-shaped, as if sharply cut off, descending toward the floor of the ulcer.

They are digested in so far as the tissue is unable to resist the peptic power of the gastric juice. In older ulcers a reactive inflammation sets in at the periphery, and leads to the formation of a calloused margin. Here the supporting fibers between the resisting tubules are thickened, and partially formed into oblique layers. The glandular epithelium, in so far as it is still present in the fundus of the glandular nests, has suffered a peculiar change. In place of the lab-cells, cubical or cylindrical epithelia have appeared. These masses are contracted so that they are separated from the membrana propria and from one another. Their nuclei cannot be recognized by staining, their contents have a friable, light, glassy appearance like that of hyaline degeneration. The individual tubules show cystic degeneration. The submucosa is permeated by a profuse small cell infiltration and a strong vascular net. The layers of the muscularis ar separated by a fibrillary, intermediate tissue, consisting of meshes torn asunder, some of which have been destroyed. We see, therefore, that th entire necrotic process upon borders and floor is surrounded by a zon of irritation, which in its further development leads to true cicatrization in that the floor of the ulcer constantly becomes adherent to the laye of tissue beneath, and the mucous membrane of the border of the ulce retracts into its floor.

In the interstices of the glands, not only in the immediate vicinity of the ulcer, but also in those more distant, the mucosa invariably shows massive small cell infiltration, but nothing peculiar to ulcer, only what is found in all inflammatory irritations of the mucous membrane from a mild catarrh to an acute phlegmonous gastritis. I have rarely examined a gastric mucous membrane, no matter whether the patient suffered from a gastric affection or some other chronic disease, without discovering here and there at the base of the mucosa disseminated foci of small cell infiltration (not lymph-follicles by any means!) which had separated the glandular tubules from each other. In fact, the ulcer subsequently produces an irritation in the surrounding areas of the mucous membrane which to a varying extent and intensity implicates this tissue.

The outcome of the necrotic process is especially interesting. We must differentiate between the following conditions:

1. **Cicatrization.**—Here there is a marked difference from the cicatrization produced artificially in the animal. For while these cicatrices, as Cohnheim reports, and as I also found, healed by the restitution of the normal mucous membrane tissue, in man a fibrous cicatricial tissue with a central depression and the well known tendency to contract develops. Thus, striated cicatrices form and produce a distortion of the gastric wall, particularly when the cicatrix is fixed in position by a preceding adhesion with neighboring organs. Girdle-like constrictions form which give the organ an hour-glass or a cucumber shape. Peculiar cicatricial bands or bridges develop, and form a many-celled sac, of which Cruveilhier gives an excellent illustration in his Atlas.

2. **Progressive Corrosive Necrosis.**—This process, provided cicatrization does not occur, continues as long as peptic gastric juice is formed, and finally terminates spontaneously, i. e., by the complications which arise. These are:

(a) *Erosion of Vessels.*—According to the seat of the ulcer and its extent and depth, sometimes smaller, sometimes larger, lumina of the vessels are eroded. The slight tendency to permanent thrombus formation is characteristic, and is probably due to the corrosive effect of the gastric secretion. Among the larger vessels the pancreatic artery, the gastric artery, and the renal arteries, or the corresponding veins, are attacked.

Severe hemorrhages follow which are only terminated by thrombotic coagulation of the blood. If the thrombus is situated in the splenic vein it may produce acute enlargement of the spleen.

(b) *Adhesions to Neighboring Organs and Perforation.*—If the necrosis extends to the serosa, either a reactive inflammation and adhesion to neighboring organs with consequent implication of these, a perigastritis, or, if this be impossible, direct perforation into the abdominal cavity, is produced. Through the corresponding intermediate tissues secondary perforations may take place into the pleural and cardiac cavities. All of the neighboring organs, the liver, the gall-bladder, the pancreas, the the diaphragm, the heart, the lungs, and the intestines, accordin seat of the ulcer, are exposed to this danger. Occasionally there a sions to all of the neighboring organs in the abdominal cavity an actual perforation, such a condition having been described by

Finally the tubercular and syphilitic ulcers are to be mentio

Tubercular ulcers, which, so far, have always been found in c tion with tubercular lesions of others organs, are characterized thickened, infiltrated, wall-like edges. Their floor is usually g and of a light yellow color. They are pale and, as in the cases o ger, rise sharply from the darkly stained surrounding areas.

In the margin and upon the floor we find tubercular nodu characteristic giant cells. The ulcer may be solitary or multi only affect the mucous membrane and mucosa, or also the m Microscopic examination shows the well known tuberculous f numerous tubercle bacilli. In some cases (Litten) the serosa for floor of the ulcer was permeated by miliary nodules. In Litten's ulcer was quite large, 4.2 by 3.3 ccm., its border sharp and partly swollen and hemorrhagically infiltrated, the remaining tract free from tuberculous ulcerations, but these were presen larynx, bronchi and lungs. Similar cases have been reported by Balzer, by Giles Saburin, and two by Eppinger. An excellent d of a tubercular ulcer is given by J. H. Musser. The case was negro, 44 years of age, suffering from pulmonary tuberculosis defined dyspeptic symptoms. At the autopsy a gastric ulcer w 1½ by 3½ inches, and undoubtedly of tuberculous nature. Man tubercles and much caseous material were found upon its floor, a cles in the neighboring submucosa. In this caseous mass wer bacilli. The case of Bar-Bacci is similar: Isolated, tuberculous u of the stomach with primary, pharyngeal tuberculosis.

Musser mentions a few similar cases, mostly in children, also tubercle bacilli were found in the ulcers. Evidently thes are rare, for in 567 cases of tuberculosis of the intestine Eisenh gastric tuberculosis in only one case, and in nearly 2,000 ar tubercular individuals Simmonds found only eight cases. Ma study of gastric disturbances in pulmonary phthisis, mentions examples, and these are somewhat doubtful.

In children the affection appears to be more frequent, for in cular children O. Müller and Hecker found 8 cases of tubercul stomach, and Biedert collected 41 cases of which one-third o children. These ulcerations do not all belong to the type of th ulcer; on the contrary, some are due to true tuberculous foci o with a caseous decomposition of the tubercle, or, at all event combination with the corrosive action of the gastric juice up crosed tissue elements. Their point of preference is the pyloric region, and their development may certainly in most cases be attributed to the deglutition of sputum containing bacilli. Occasionally they may have formed upon a hematogenous and lymphogenous base. Such ulcers may also perforate into the abdominal cavity and produce the symptoms of perforative peritonitis. Kundrat, Paulicki, and Struppler have reported typical cases of this kind.

Syphilitic ulcer has no special anatomical characteristics. In the majority of cases that have been accurately observed no opinion is expressed as to whether we are dealing with a primary lesion or with a decomposed gumma. The syphilitic nature of the ulceration has been recognized (Luxenburg and Zawadzki) only by the presence of an endarteritis of the gastric vessels. Engel believes that 10 per cent., and Lang that even 20 per cent., of all ulcers are of a syphilitic nature, but this seems a gross exaggeration. The syphilitic nature of the ulcer is favored by the fact that typical symptoms occur simultaneously with the secondary symptoms of syphilis, and are cured by antisyphilitic treatment (Cullevrier, Schwimmer, Leudet, Murchison, Hayem, Gaillard, Cesaris Demel).

The fact that a *carcinomatous neoplasm* may develop upon the floor of an ulcer, preferably one situated near the pylorus, requires especial mention. This was emphasized by the Vienna School, and proven beyond doubt by Hauser. In my opinion this fact is not affected by the unsuccessful attempt of P. Hirschfeld to prove by statistics that cancer of the stomach occurs independently of the presence or absence of gastric ulcer. Everything can be proven by statistics, but these do not militate against the force of direct observation, and the latter will convince all who have had considerable practical experience (instead of having seen but a few cases) that, in many instances of gastric ulcer, particularly those seated at the pylorus, there is a gradual but uninterrupted transition of the ulcer into cancer. Borrmann, in his excellent monograph upon gastric cancer, in which he differs from Hauser, does not deny this change, as Hirschfeld erroneously assumed. In their ideas of the propagation of the neoplasm into the tissue, these investigators are wide asunder. Clinically, however, these are the cases in which hydrochloric acid production and the peptic function of the stomach continue for a long time, although they gradually decrease toward the norm—cases which, upon the basis of chemism, have been diagnosticated as benign tumor of the pylorus, while operation or autopsy shows a more or less carcinomatously degenerated old ulcer.

It is always difficult to quote statistics in regard to the *frequency* with which definite diseases occur and their peculiarities; rather, to utilize the so-called statistics for nosological and diagnostic purposes. We deal with different classes of patients and different conditions, which to a great extent depend upon unavoidable circumstances. If, however, in the concrete case we attempt to base our opinion upon statistics, we may not know whether the case is typical or exceptional. It is true that certain circumstances, that is experiences, are so positive and invariable that they require no statistical proof. Among these is the well known fact that women suffer more frequently from gastric ulcer than men, but the reports of authors differ widely as to the exact proportion. Joslin gives the ratio as 1:5, Rheinwald as 1:1.2. Both authors base their figures upon comparatively few cases, respectively 190 and 187, while in 3,114 cases Murrell found a proportion of 1:2.1 and 1:3.4. All authors agree that the age in which most are affected is the third decade, i. e., the time between the twentieth and thirtieth years of life. Among 2,164 cases Murrell found 393 between the twentieth and thirtieth, and 265 between the thirtieth and fortieth, years of life. Joslin found the average age in men as 37, in women 27. In Rheinwald's cases 9 males and 31 females between sixteen and twenty years of age, and 19 males and 22 females between twenty-one and twenty-five years of age, were attacked.

It is well known that the proportion in cancer of the stomach is just the reverse, i. e., that more men than women are attacked, and that in later life those between the fiftieth and sixtieth years are most liable; therefore, the comparative youth of the patient in a doubtful diagnosis of ulcer would be of some weight if we had not recently learned by surprisingly numerous experiences that youth is not exempt from cancer. The youngest case of cancer of the stomach that I have observed occurred at sixteen, the oldest at sixty-five years of age. It is true that in the former case carcinoma had developed from an ulcer.

SYMPTOMS

The symptoms of gastric ulcer which authors have described as belonging to the so-called normal type need not be dwelt upon; only the most prominent will be referred to.

Our discussion will be chiefly devoted to the manifold deviations which often make the so-called classical clinical picture appear quite differently from that described in the text-books.

The symptom-complex of ulcus ventriculi may be divided into the following groups:

1. Cases in which the irritative symptoms which result from hemorrhagic erosion or the corrosion of larger or smaller areas of the mucous membrane predominate, and there are no complications.
2. Cases in which the previously mentioned irritative symptoms and hemorrhage simultaneously appear.
3. Cases with symptoms of irritation and perforation.
4. Cases which run a latent course up to the time of death from hemorrhage or perforation.
5. Cases which present the sequelæ of ulcer.

When we remember merely the fact that in these groups the symptoms may be variously combined, an ever-changing clinical picture is presented.

As is well known, we usually diagnosticate gastric ulcer when the classical triad of symptoms is present: Hematemesis which can be definitely traced to the stomach; typical, and usually sharply localized, pain occurring at regular intervals after the ingestion of food; and the increase of free hydrochloric acid in the gastric contents.

From this it is evident that gastric ulcer can only be diagnosticated with certainty in advanced stages, and can only then be recognized if the previously mentioned symptoms occur in combination. *This, however, in my experience is by no means invariably the case, and I might even say that such cases are exceptional.*

As the nerves exposed in the ulcerated surface are irritated by the ingesta and thus the pain is produced, and the muscular contractions which arise in consequence of the irritation may also cause painful sensations, *gastralgia* soon forms an important feature of the clinical picture.

Early in the affection the pain becomes localized in a definite area, usually the seat of the ulcer, and mostly in the pit of the stomach. The boring, sharply defined pain which frequently extends antero-posteriorly is characteristic. It usually increases upon pressure, and its intensity may be determined by the so-called algesimeter which is employed by nerve specialists, without the diagnosis becoming any clearer or any points of support being gained as to the localization (Boas). Women cannot lace, and men cannot wear a firm belt; only exceptionally is the pain ameliorated by pressure. It is obvious that the pain is most intense when the raw, ulcerated surface is exposed to great mechanical irritation. First, and most frequently, that after eating, either because the ingested food directly irritates or by its weight distends the gastric wall, that is, causes contraction and spasm, especially if the ulcer is located at the pylorus. This is relieved as soon as the stomach rids itself of its contents; as, however, small or coarse particles of food are occasionally caught in the grooves of the ulcer and may remain there, it is not remarkable that the organ to a certain extent reacts to these irritative bodies, and repeatedly makes an effort to expel them. Thus spasmodic contractions, *pylorospasms*, which may last for days or may recur at short intervals, arise and greatly weaken the patient by their duration and the suffering they cause.

These are generated in highly developed processes of stenosis of the pylorus or of great atony of the muscles of the stomach by the retention of acid products of secretion, with the starches which are feebly or not at all digested. Distention of the stomach from gas and irritation of the nerves by the continuous ulcerative process produce gastralgia, which must be looked upon rather as a reflex effect when cold and psychical stimulation produce gastralgic attacks, or the pains increase in severity before the menstrual period, and lessen when it appears.

These, however, are not the only causes. In patients with peptic ulcer I have repeatedly seen severe gastralgia after a cold drink or a spoonful of hot soup, a swallow of too hot tea or any similar thing; in these instances the previously mentioned factors were out of the question, but a thermic irritation was the cause. In my experience too hot ingesta is a cause much more rarely than cold, perhaps for the reason that with the former, the mouth and pharynx to a certain extent act as gourds, the gastric mucous membrane is more tolerant to high temperatures than to low ones, and also because less is consumed of the former than of the latter.

Gastralgia accounts for the fact that these spasmodic pains usually come on suddenly and with great intensity, and that they disappear almost as rapidly as they occur, and the patient quickly returns to nearly a normal condition, while a gradual increase to a paroxysmal height is rarely heard of.

The occasional occurrence of cutaneous hyperesthesia and anesthesia, as was observed by Traube, is peculiar, and is attributable to central irradiation. Attacks of angina pectoris, of intercostal neuralgia, of neuralgia of the left brachial plexus, of sensations of pain in the right shoulder (Brinton) and in the arms and legs (Müller) appear. Pressure points are noted upon the back, and along the vertebral column, corresponding to the posterior roots of the lower thoracic and upper lumbar intercostal nerves, or a spontaneous pain, or one after pressure, appears in the spinous processes.

In opposition to some recent investigators, particularly Pariser, I maintain that these pain and pressure points upon the posterior walls of the thorax *are by no means invariable*. On the contrary, in my patients they were often absent, and, like the author mentioned above, I should be unwilling to base a diagnosis, or the permanency of the cure of an ulcer, upon their presence or absence. As the demonstration of this phenomenon is very simple, errors can hardly result from the method.

At this point *tetany* must be mentioned, which, however, is but slightly connected with gastric ulcer, being not an immediate but only a remote sequel. This peculiar symptom-complex is often attributed to a deficiency of water in the organism, and is characterized by the hyperirritability of certain peripheral nerves, above all, the facial nerve and those which innervate the extensors of the extremities; among other conditions it occurs in gastrectasis following ulcer of the pylorus, and gives rise to stenosis.

Gastric dilatation which, in its course, leads to stasis of the gastric contents and a deficiency of water in the organism, may, however, be due to various causes. Hence, tetany does not clinically belong to the picture of gastric ulcer, but is only a complication which occurs in a slight number of cases. At this point I cannot refrain from expressing my belief that deficiency of water in the organism, brought forth so prominently by Kussmaul's adherents, is the causative factor in only a small number of cases. Other authors, as well as I, have observed cases of tetany in which there were no signs of gastrectasis and its consequences, but the cause was to be sought for in the increased reflex irritability due to abnormal decomposition of the ingesta and the formation of toxins thereby.

In nearly three-fourths of all cases of gastric disease, there is an "*epigastric pressure point*," i. e., an area in the scrobiculus cordis sensitive to pressure, and this is also present in most patients suffering from ulcer. That this has nothing in common with the localization of the ulcer is a well known fact and experience. To be distinctly differentiated from it is a point lower down and to the left which upon pressure also very frequently, but not so often as the first mentioned one, is sensitive to pressure, and in its position corresponds to the solar plexus. In fact in most persons, with and without ulcer, a decided sensitiveness to pressure may be observed in this area, provided the pressure is exerted obliquely toward the median line, and thereby the solar plexus is forced toward the bony vertebral column. That in thin persons with rigid abdominal walls a distinct epigastric pulsation is felt in this area hardly requires mention.

Only rarely is the accompanying *gastric catarrh* so prominent that actual anorexia, a pappy taste, eructations, a disagreeable odor to the breath, and a *coated tongue* are observed. On the contrary, the latter organ is usually of fair appearance, smooth, moist, and distinctly red, so that the findings just mentioned in patients with severe, colicky, gastric disturbance should at once awaken our suspicions of ulcer. What of the *chemical processes* going on in the stomach?

Here we are forced to touch upon the question so often discussed whether it is permissible, with a certain or suspected ulcer, to introduce the sound or the stomach-tube for diagnostic or therapeutic purposes. Absolutely yes! In many hundreds of patients with ulcer I have introduced the stomach-tube, or have had it introduced, thousands of times, and have *never* seen deleterious effects.

This is maintained by all authors of great experience. Of course, this manipulation is not to be undertaken *immediately* after a profuse hemorrhage. But we shall see later on that during decided bleeding it is very beneficial to wash out the stomach with ice-water.

That in a majority of the cases the chemism is not decreased is evident from the fair appetite of patients with ulcer. Yet the early view that an increased production of hydrochloric acid is a necessary or, at least, an almost invariable accompaniment of gastric ulcer can no longer be maintained, being contrary to the comprehensive observations of the last few years. In my cases I found hyperacidity in only 34.1 per cent., normal acidity in 56.8 per cent. and subacidity in 9 per cent.; I must admit that these results surprised me greatly. It must be remarked, however, that in the last mentioned group there were five cases in which the acidity on repeated investigations was found to be either 14 per cent. or 24 per cent.,

while the presence of a typical gastric ulcer without carcinomato
eration was made certain by an operation and the subsequent mi
investigation of a resected portion of the tissue. It is true th
other cases of this category we were dealing with persons in whor
or less advanced degeneration was either assumed or was determi
tively by operation.

Rheinwald tested the chemistry of the stomach in 66 cases, a
hydrochloric acid in 84.5 per cent., hyperchlorhydria in 65.5 per
absence of hydrochloric acid in 7 per cent., lactic acid 5 times.
quency of non-increased, and even subacid, values has also been
other authors.

In 38 cases of ulcer Schneider found hyperacidity in only
per cent. The reason he assigns for this is that simultaneously
tions of various kinds arose, such as ectatic conditions, chronic p
more or less severe anemia, etc.

Köhler, who observed the cases in Gerhardt's Clinic from 189
found among 165 tests for hydrochloric acid 65 per cent. of pos
34.7 per cent. of negative results. It is true the extreme
of lactic acid, namely, 33.3 per cent., forces us to conclud
many of his cases there was already well advanced carcinom
generation.

According to these experiences, the opinion that all gastric ulc
the time of their existence cause a typical increase of acidity, n
events be rejected as incorrect.

On the contrary, here—as in many other points in the di
gastric disease—we must be very cautious in utilizing the
findings. Increased acidity favors ulcer, its decrease does not
exclude it.

In fact, upon repeated examinations the values for acidit
cided variations. In 35 cases, in which I was not content wi
test but made repeated examinations, sometimes six or seven,
in some respects differed widely from each other. Thus, in a w
25, who several times suffered from hematemesis, and in whom
contents after a test breakfast frequently showed small quantiti
the values for acidity varied between 28 and 44; in a patie
between 36 and 78; in a man with repeated hematemesis, an
ished from perforation, between 38 and 54; in another patien
resection was performed for ulcer at the pylorus, between 29
Treatment, and particularly the diet, influences these variatio
are especially important because these are usually the cases i
resort to the stomach-tube to obtain a positive finding, if possi
also states that in some cases of undoubted ulcer of the stoma
of tests are apt to be negative until free hydrochloric acid is
large quantity, and sometimes no hydrochloric acid can be

any tests. *Riegel's statement that hyperacidity is a constant sign in ulcer of the stomach must, therefore, be accepted with limitations.*

Gluczinsky has called attention to the fact that chemism changes when a correct diet is instituted, that is to say, changes with the food. For example, in the first week on a mixed diet an acidity value of 30 (hydrochloric acid) was found after a test breakfast, in the second week upon a milk diet 8, in the third week upon mixed diet 25, and in the fourth week upon a milk diet 5. In the cases which I cited it is unnecessary to consider this circumstance; for, during the entire time the patients were under observation, they were kept upon the same bland diet.

In a certain group of cases, however, an increase in the excretion of hydrochloric acid is unquestionably present, and these are the cases designated as *gastrosuccorrhea* or *hypersecretion* (parasecretion, Ewald). Formerly relegated by many authors to the true neuroses, they are now regarded as the consequences of ulcer running a latent course. Soupault operated upon 28 cases of typical gastrosuccorrhea, and in each instance found an ulcer of the pylorus. In 48 cases of gastrosuccorrhea Mathieu and Laboulais found hematemesis or melena six times, and eleven times very severe pains were referred to spasm of the pylorus. Gluczinsky attributes all cases of gastrosuccorrhea to ulcer with stenosis of the pylorus. This, in my opinion, is to act without discrimination. The outpouring of gastric juice is occasionally irregular and unsteady. It is unreasonable to assume in each case a newly formed ulcer, but in all of these instances we must consider nervous irritation as a cause, as well as the fact that these cases cannot be strictly separated into "*retention secretion*" and "*hypersecretion*," nor do they belong exclusively to one or the other category. Every one of extensive experience knows that transitions occur, and that the same case may at certain times show the characteristics of a pure neurosis, and at other times may present the picture of deranged stasis. Therefore, explicit descriptions to show that either the one or the other condition was alone decisive and causative appear to me to be quite superfluous. Yet this is now a secondary question. I desire only to show that certain cases of ulcer positively present increased secretion of hydrochloric acid, and, disregarding the limitations mentioned above, the latter, i.e., hyperchlorhydria, is unquestionably of great diagnostic value.

I go a step further, and maintain that in all cases in which the course points with more or less likelihood to gastric ulcer, the chemism of the stomach, however, showing insufficiency of hydrochloric acid secretion and of peptic digestion, the view of a carcinomatous transition of the ulcer is well founded. This is particularly true in young persons. In this respect I have recently seen a most typical case: A woman, aged 26, had a large, freely movable tumor situated upon the greater curvature and toward the pylorus, the mucous gastric contents showing undigested remains of a roll but without free hydrochloric acid. Operation revealed two flat ulcers the size of a twenty-five cent piece, one close to the pylorus, the other toward the fundus, in the surroundings of which the submucosa, the muscularis and serosa were markedly thickened. The floor of the ulcers was almost smooth, of a light flesh color, the margin was slightly raised, but not undermining the neighboring, greatly swollen mucous membrane. Microscopical investigation showed a carcinomatous neoplasm extending into the muscularis, which only in quite isolated areas had left some glandular tissue. In this case the history revealed no former symptoms of ulcer, but the age of the patient was against a primary carcinoma. In other similar cases, however, typical signs of an old ulcer could be gleaned from the history.

The hemorrhages are either from small vessels and slight in amount, and then are only accidentally observed in the form of fine hemorrhagic streaks when freshly admixed with the vomited material, or they are seen as reddish-brown, granular masses after the gastric juice has for some time acted upon the accumulated blood. Upon superficial examination small hemorrhages of this kind may be entirely overlooked, for the blood is not vomited at all, but passes into the intestine, and is there so decomposed that the appearance of the feces is not decidedly altered. Slight admixture of blood to the gastric contents does not give it a characteristic appearance, but often only a dirty gray color, which does not at once awaken the suspicion of hemorrhage. On the other hand, it need hardly be stated that cocoa, chocolate, strawberry wine, bilberry wine, and the like may lead to errors upon superficial examination. In such cases, as Schmauss has shown, we must occasionally make microscopic, spectroscopic, or chemical examinations in order to detect the presence of the smallest quantities of hemoglobin or of blood in the gastric contents or in the feces, and to recognize them as the cause of so-called essential anemia.

Occult Hemorrhage.—In latent gastric ulcer Rossel, and subsequently Boas and I, repeatedly made the diagnosis of ulcer from the proof of "*occult*" blood in the feces, naturally, when other sources of hemorrhage were excluded, and the food administered prior to the examination did not contain large amounts of blood.

The *chemical examination* of the feces for blood is best made either with the resin of guaiac or with aloin. In either case a quantity of feces about the size of a hazelnut is rubbed up with water (or a corresponding quantity of fluid feces is taken), and, after the addition of some glacial acetic acid, is shaken up in a test-tube with ether (the previous removal of fat from the feces by shaking with ether is proper, but is by no means always necessary). The hemoglobin which may be present is changed into hematin (methemoglobin) by the acetic acid, and taken up by the ether. To the clear, over-lying ether which is poured off resin of guaiac which has been dissolved in alcohol is added (or the dry powder which is readily soluble in ether), and finally about a cubic centimeter of the resin of turpentine or Hühnerfeld's reagent.[1] When larger quantities of blood are present, a blue color at once appears; when smaller quantities, this change takes place after a few minutes. The test is made in the same way with aloin, of which a freshly prepared alcoholic solution should be used. In a few minutes the ethereal mixture changes to a beautiful, cherry-red color, and the hemoglobin forms a uniform layer of precipitate at the bottom of the test-tube. According to Boas, the test may be made in the same way as for the reaction of indican; the addition of a few drops of chloroform will make the reaction more distinct, but in my opinion this is superfluous. No matter how the reaction for blood is produced, in those cases which present no other characteristic sign of gastric ulcer the difficulty will be to prove that the blood found is actually from the stomach and not from other portions of the digestive tract. It is self-evident that no blood or hemoglobin should be given with the food.

The *microscopic proof* of small quantities of blood in the feces is always very difficult, because the blood-corpuscles in their migration through the intestine are usually so distorted that they lose their characteristics. In profuse hemorrhages there is no danger of this, but in lesser ones, especially when mercurial preparations or those containing sulphur have been administered, it must be considered.

Copious hemorrhages presuppose the erosion of a large vessel, and they act upon the stomach like an emetic, so that this organ rids itself of its contents. Many patients have distinct premonitory symptoms, flashes of heat, epigastric pulsation, a sense of fulness in the gastric region, great, and apparently groundless, internal disquietude which to a great degree produces depression and anxiety. In other patients the only preceding symptoms are an unusual feeling of illness and complete loss of appetite. The time which the blood remains in the stomach varies greatly, and this also changes the appearance of the vomited material. Sometimes the coagulated blood is light red and lumpy, at other times it forms dark red masses, and sometimes, in a minority of cases, the vomited material resembles coffee-grounds. Large quantities of blood in the vomited material may even be noted with the naked eye, certainly by the aid of the microscope, perhaps by the spectroscope, or by the various chemical tests for blood. As mentioned above, a portion of the blood finds its way into the intestines. If a profuse hemorrhage has occurred, or if the ulcer is in the duodenum, the evacuations have a tarry appearance, and consist of very offensive masses in which the presence of blood may be detected.

In most cases the hemorrhages occur suddenly without an assignable cause. Occasionally they are preceded by circumstances either of a psychic or a physical nature which accelerate the cardiac action, such as joy, fright, unusual exertion, external trauma (blow, fall, pressure, etc.), or strain upon evacuation. Improper food, for example, hard tendir fibrous vegetables, such as beans or asparagus, may cause hemo direct mechanical lesion. One of my patients, who up to that never vomited blood, had a severe hemorrhage after taking, on of Cohnheim, 200 grams of oil by the mouth to relieve pylor Nausea, retching, and hematemesis were the consequences of thi medication which is greatly praised by its author.

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[1] Acid. acetic., Aq. destill. āā. 2.0, Ol. Terebinth., Spirit. dilut. āā. 100.0.

The frequency of hematemesis is reckoned at 50 per cent., b too high rather than too low. Brinton gave 29 per cent., Witte hagen found it 100 times in 339 cases (= 29.4 per cent.), Gerha in 47 per cent. of his cases, so that we may safely assume that half of the patients do not suffer from hematemesis. Among v. Leube found it in 46 per cent. I observed it in 54.5 per cent cases Joslin found no less than 81 per cent. showing gastric her from which we may see how unreliable and how dependent upon conditions these so-called statistics are.

In an interesting compilation based upon the records of Guy' from 1870 to 1890, W. Charles Hood reports that in the majori of gastric hemorrhage in the course of ulcer occurring in pati 30 years of age women are chiefly affected, and that during thi life fatal hemorrhage is extremely rare. Of 66 cases of this ki under thirty, among these only 2 men. On the other hand 11 men among 21 cases between thirty and forty years of age. ered. Seven other cases in which death occurred immediatel hemorrhage were all over 30 years of age, among them 4 wom ively 33, 35, 50 and 53 years of age. Although we can har that the prognosis of gastric hemorrhage changes particularl critical age of thirty in women, nevertheless this report in gastric hemorrhages in younger women present a less unfavo nosis, since a conspicuous improvement, the cessation of the symptoms and regeneration of the blood, is often observed af event. In one of my cases the number of erythrocytes within after the hemorrhage increased from 1,900,000 to 3,040,0 amount of hemoglobin from 31 per cent. to 51 per cent. Sim tions have been repeatedly made.

The hemorrhagic masses from a bleeding peptic ulcer ar specific elements, and the blood-corpuscles preponderate to su that the cellular elements of the gastric mucous membrane are or do not appear at all.

When a copious hemorrhage has once occurred the danger tion is always present and hangs, like the sword of Damoc patient's head in a two-fold manner. In the first place, at b in the course of a day, or even several times a day, or, per intervals during a week, hemorrhages repeatedly occur. W assume that we are dealing with relapses from the same vessel which was first eroded. Secondly, after longer intervals, months or even years, hematemesis may be renewed, and then, in consequence of the tendency of the individual to hemorrhages of this kind, repetitions are likely. Occasionally it appears as though the thrombus formed is not adherent, and that it readily sloughs off when the cardiac action becomes stronger, as under normal conditions. Twice I saw a hemorrhage occur after a long interval when the patient, from a mistaken idea that it would be beneficial, took strong alcoholic liquors, although only in small quantity.

Slight hemorrhages, aside from their psychical effect, have no influence upon the condition of the patient; copious hemorrhages, particularly when in rapid succession, lead to extreme anemia and its consequences. Wax-like pallor of the skin, a small frequent pulse, slight fever, tinnitus aurium and vertigo, loss of consciousness, transitory mild delirium, and total anorexia follow. Subsultus tendinum and spasms in the extremity like those of cholera have been observed. Nevertheless, the patients recover with comparative rapidity, and under suitable treatment the lost strength is regained. Edema of the extremities, which is observed particularly in the evening if the patient has been upon his feet during the day, and amaurosis which sometimes occurs immediately, perhaps some time after the hemorrhage (but which, according to Fries, occurs in 65.5 per cent. of all hemorrhages in the intestinal tract), show an internal relation to hematemesis which as yet has not been clearly demonstrated. As has already been stated, cases of gastric hemorrhage with fatal outcome, particularly in youth, are comparatively rare.

Here also the reports of authors differ widely, and the results vary between 8 per cent. (Rodman) to 0.8 per cent. (Bramwell). My own statistics, based upon 360 cases observed in the hospital, give 1.2 per cent.

Usually death is due to the rupture of the ulcer and corrosion of the splenic or pancreatic artery, the portal vein, or the left heart, as will soon be described. A case of chronic gastric ulcer with fatal hemorrhage from an eroded left renal vein has lately been reported by A. Markel.

A small aneurysm of the coronary artery was the cause of death in a case described by Powell. The ulcer was situated in the lesser curvature close to the cardia; in its center was an aneurysm about the size of a pea which had ruptured; and by profuse hemorrhage had caused death in a few moments. A similar case was described by Sachs. Here a small artery of the submucosa was implicated.

The hemorrhages which take place by the perforation of an aortic aneurysm into the stomach or into the esophagus are of a more indirect nature. Gastric hemorrhages have also been observed (Naunyn) in cholelithiasis. Minkowski reported a remarkable case at the Congress of Internal Medicine in 1902. It was that of a small aneurysm at the arch of the aorta which had ruptured through the walls of the esophagus.

Thence the blood found its way to the cardia and to the floor of an ulcer in the stomach.

Any of these hemorrhages may occur without preceding symptoms of an ulcer of the stomach. Fatal hemorrhages are usually preceded by indistinct signs of a severe disease; in other cases, however, the hemorrhage may occur suddenly like lightning from a clear sky, attacking a person apparently in the best of health, and bringing about a fatal termination.

At this point we must call attention to other forms of "gastric hemorrhages," which, because less familiar, may tend to grave diagnostic errors.

First, are the *hemorrhages from varicose veins of the esophagus.* Here the condition is such that occlusion of the hepatic circulation takes place, and the blood in the portal vein attempts to force its way through the venous plexus formed by the combination of the gastric veins with the esophageal veins. The blood then takes a retrogressive course, and, as the esophageal veins cannot withstand the force of such large amounts of blood, they become enormously dilated. The least resistant areas dilate and form varices, which, with the great pressure of the blood, readily rupture and cause profuse hemorrhage. The blood at first flows into the stomach and is vomited as a "gastric hemorrhage." Only a thorough investigation and the consideration of the just mentioned possibility will furnish a clue to the true situation. Frequently this is revealed only at the autopsy. I have seen a great number of cases of this kind in which grave errors were made. Only recently a gentleman from the provinces was sent to me with the diagnosis "hematemesis from an ulcer of the stomach"; examination revealed a decided enlargement of the spleen and a hard, enlarged liver. In the gastric contents there was a marked decrease of free HCl; no enlarged glands. The patient had twice suffered from a decided gastric hemorrhage which had appeared without prodromes. Typical symptoms of ulcer were never present. Under these circumstances a diagnosis was made of hepatic cirrhosis with hemorrhage from the esophagus, and this was soon confirmed by the development of marked ascites.

In this group we must include the hemorrhages *from varices of the gastric mucous membrane* (Sachs, Letulle, A. Cohn), which often recur in close succession, and "are combined with venous stasis phenomena of the abdominal organs, often with hepatic cirrhosis, and almost invariably with decided enlargement of the spleen."

In regard to the splenic tumor in these cases, it must be borne in mind that, under some circumstances, as has been previously stated, thrombosis of the splenic vein from a corroding ulcer of the stomach may cause acute enlargement of the spleen. If the thrombus be simultaneously affected, and if, in consequence, a septic fever develops, the diagnostic perplexity is less great.

Another form of gastric hemorrhage not originating from ulcer is the so-called "*parenchymatous gastric hemorrhage.*" There is also a group of hemorrhages which are not limited to the stomach, but attack the entire intestinal tract, and occur as the consequence of venous stasis in diseases of the heart, the liver, and the lungs, particularly in thrombosis of the portal veins, and in infectious diseases, such as enteric fever, yellow fever, cholera, etc. As a rule, they do not lead to typical hematemesis, and scarcely ever simulate gastric ulcer. The condition is quite different when the bleeding appears suddenly, as a single or repeated attack of hemorrhagic vomiting, and the most minute investigation fails to reveal the source of the hemorrhage. As many cases of gastric ulcer run a latent course prior to the appearance of severe hematemesis or melena, we will not err in such cases, after excluding the previously mentioned causes of hemorrhage—here, in my experience, hepatic cirrhosis takes precedence—if we assume a gastric ulcer, and in the further course this view is confirmed by the fact that, soon or late, the classical picture of gastric ulcer develops. Now and then, however, the course is exactly the opposite of this. The hemorrhage soon ceases; for a few days or weeks, although great debility remains, the patient feels perfectly well, and shows no gastric symptoms. On the other hand, repeated hemorrhages may lead to collapse and death. At the autopsy, except for the occasional but by no means invariable superficial erosions of the mucosa, no pathological changes are found which are either directly or indirectly related to the hemorrhages. There are quite a number of such cases in literature—I have seen five such, some of which were published by Reichard—in which the most careful microscopic examination did not show the origin of the bleeding. In a case of Hampeln's, a few erosions of the mucosa were found near the pylorus and the lesser curvature, but, upon injecting the gastric artery, no fluid passed through the eroded areas of the mucosa. In one of my cases the hemorrhage was due to extreme irritation of the gastric mucous membrane, the drinking of a hot infusion of red wine, chamomile, thuja, and various spices. Under some circumstances nothing remains but the assumption of a special permeability of the vessels which are unable to resist the local hyperemia. A case described by Hirschfeld occurred in an old, cachectic woman in whom no change except a very marked arteriosclerosis of the vascular system could be found. Minkowski reports a case of amyloid degeneration of the heart and gastrointestinal vessels without a similar change in the large glandular organs (liver, spleen, kidneys) leading to parenchymatous gastric hemorrhage.

Menstrual gastric hemorrhages, and those occurring *vicariously* in place of menstruation which sometimes may appear intermediately between two periods, usually belong to the group of so-called occult hemorrhages. Actual hematemesis is not observed. On the contrary, the blood is discovered accidentally, or is found while evacuating the gastric contents (Kuttner). Therefore, this condition may very readily be overlooked;

nevertheless, it exists, and v. Schrötter, Sr., only placed his t
mental ignorance on record when he chose to designate my repor
Congress of Internal Medicine (1902) as a canard ("Räubergeschicl
Whether simple stenosis of the pylorus may be the cause of the hem
as was assumed by Lambotte and subsequently by Moser (retention
tric contents, venous hyperemia of the mucous membrane, and n
contractions) appears to me very doubtful when we contrast this
numerous cases of pyloric stenosis without hemorrhage. The c
ported by Lambotte certainly do not permit us to exclude the po
of ulcer. At this point, too, the occurrence of profuse initial hem
in gastric carcinoma must be mentioned (E. Mey). In such c
assumption is obvious that an ulcer originally existed which mu
undergone carcinomatous degeneration. In the 4 autopsy reports
nicated by Mey, the nodular character of the neoplasm favors a
carcinoma, for, in the transition of an ulcer into carcinoma an inf
of the tissue is much more likely to occur. At all events, in su
the possibility of this must be considered.

The circumstances are different when hemorrhages occur as c
tions of severe purulent processes or marked circulatory disturban
intestinal vessels. Surgeons, for example, v. Eiselsberg, have fo
time called attention to the fact that after abdominal operations,
larly for strangulated hernia, gastric hemorrhages may occur. T
attributed to displaced thrombi from the point of operation. E
plain the condition in the following case differently:

A man, aged 47, was suddenly attacked with severe pain in the umbili
there was absolute constipation. After twenty-four hours an operation
formed, at which a diagnosis of internal incarceration was made. Prior to
tion the stomach was washed out, and large quantities of a dark, browni
were evacuated; this proved to be almost pure blood. At the operation
of the small intestine amounting in length to 1 meter and 30 cm. was f
constricted by a band reaching from the sigmoid flexure to the root of the
The constricted intestine was enormously distended, and dark bluish-re
The circulation was not reestablished in the constricted portion of th
hence it was resected. The patient lived only twenty hours after the se
tion. The autopsy revealed a greatly distended stomach; the leaflets
phragm were forced up to the fourth rib. "In the stomach countless
the mucous membrane of the size of a pin's head were found. Some of
round, others angular or elongated; in isolated cases, they extended
mucosa; at other points they were more shallow. Some contained greyis
particularly about the borders and at the base; other erosions contain
A *true vascular lesion* was nowhere to be found. The peritoneum near
region showed no abnormalities." (From the autopsy report of Dr. Oest
little muco-hemorrhagic contents were found in the intestine; the vessels
edly injected. The abdominal cavity contained about 200 c.c. of a h
serous fluid. In this case we probably find the cause and anatomica
for the hemorrhage in the altered circulation of the abdominal cavity
erosions of the mucous membrane which were apparently of an earlie

Severe hemorrhages into the stomach and the intestine occurring in the course of septic processes which affect the abdominal organs without an apparent anatomical lesion, and which cannot be attributed to the formation of thrombus, have until recently been observed only in isolated cases. Dieulafoy mentions them in perityphlitis, and regards them as the result of toxin infection of the gastric mucous membrane, the latter, in his cases, showing no ulceration in the region of the pylorus. Guyon has reported a case of sepsis in the urinary passages without any injury to the urinary organs. Such cases may present insurmountable difficulty in the diagnosis, as will be noted from a case which I recently observed.

A man, aged 37, who had been ill for four days, was awakened during the night with pain which soon spread over the entire abdomen. He stated that he had previously suffered from gastric difficulty, and that upon the second day of the disease a chill with rise in temperature to 103.2° F. had taken place; except for this there was no fever during the entire course of the disease. I found the patient with all the signs of diffuse peritonitis,—slight dulness in the right and left inguinal regions. The pains varied, being both spontaneous and upon pressure, and were said to have first appeared in the pit of the stomach. During the night the patient vomited large quantities of a blackish-brown mass containing blood. The vomiting recurred in the course of the day and also during the lavage with ice-water which was later undertaken, the water each time evacuated containing blood. The slight quantity of feces discharged from the large intestine after enemata showed no blood. Consequently, I diagnosticated a perforating gastric ulcer, and rejected the possibility of a perforative perityphlitis on account of the hemorrhagic gastric contents and the original seat of the pain in the scrobiculus cordis. The surgeon called in consultation was of the same opinion, but, on account of the advanced peritonitis, refused to operate. The man succumbed on the third day of my observation, and upon the sixth of the disease. The autopsy revealed general peritonitis starting from a perforating gangrenous appendix. The gastric mucous membrane was entirely intact. But the intestinal mucous membrane in some areas showed a bright red injection, a velvety swelling, and hemorrhagic contents were still present in the intestine itself.

Composition of the Blood.—Most authors—I mention Laache, Leichtenstern, Reinert, Osterspey (Ewald), and also recently Rencki and Dolmatow—have found the *composition of the blood* (and this quite independently of a possible hematemesis) so changed that a decided alteration in the number of red blood-corpuscles, and in the hemoglobin, and, occasionally, an increase of the leukocytes, was present. But these changes are due to secondary conditions, chlorosis, anemia, etc., and are not typical of ulcer. For well known reasons, the composition of the blood immediately after a hemorrhage will invariably be found changed, i. e., the red cells and the hemoglobin are decreased, and the leukocytes slightly increased.

Vomiting.—The next symptom of importance is vomiting.

Vomiting usually occurs after eating. It is caused by an irritation of the walls of the stomach, particularly of the exposed ulcer surface, from the food, that is, the hyperacid gastric contents. This is partly due to the fact that the strong acid causes a spasmodic contraction and cl of the pylorus, and thus not only pains but an increased and even peristaltic action of the stomach is produced. The food is often but changed, and is vomited admixed with mucus as is the case in the so- *vomitus matutinus* of alcoholics. Fungi of fermentation and other fc cellular elements, with the exception of occasional admixtures of l are either rare or absent (sarcinæ). At other times the vomitus is a fluid, of slightly greenish color, and very acid, which upon standing a pappy precipitate consisting of starch granules, cellular detritus, but a few markedly digested remains of meat. Periods occur in which is an increase of the vomiting, and the stomach absolutely rejects foo

It is quite apparent even from the external appearance of the voı particularly if it is ejected a considerable time after the ingestion of that the gastric contents are decidedly acid. Upon standing two form: The upper is a thin fluid, clear, not foaming, and contain finely granular precipitate consisting of slightly altered remains of s plant cells, and the like, in which, however, there are no, or very muscle fibers, that is, remains of meat. The test with Congo paper a strong reaction for free hydrochloric acid. As a rule spontaneous ing is not frequent. It is most prone to occur when the ulcer is si at the pylorus.

Perigastritis.—When the ulcer has extended to the serosa, and complete rupture takes place, an inflammation of the external layer serosa with the formation of a plastic exudate and perigastritis oc ally occurs. This may be limited to a definite area, or may exten adjacent parts, or may change to hard, indurated masses of exudate upon examination resemble tumors. Associated with this are loc tative phenomena, retching, and even mild fever. Distortion of th ach may take place and, under some circumstances, it may be very or even impossible to recognize the true nature of these conditions. their character, they are insusceptible to internal treatment, and permanent source of perplexity. (See also p. 185 in the descrip cicatrization.)

Perforation.—A severe complication of the disease and, perhaps clinical picture is produced by the *rupture of the ulcer and the imp of neighboring organs.* When the peptic process reaches the extern of the gastric wall and attacks one of the neighboring organs, occasionally manifested by a localized sensation of pain in the re the organ attacked. As a rule, however, it runs its course with external sign, so that only when we test the disturbance of functio organ in question can we recognize that it has been implicated process. As previously mentioned, there may be hemorrhages f larger vascular trunks.

The various conditions which arise for consideration may be reviewed if we remember the topography of the organs surrounding the stomach. A most interesting complication is a rupture through the diaphragm and pericardium into the left heart followed by pneumopericarditis, also into the mediastinum with emphysema of the external skin and the accumulation of combustible gas. West describes a preparation in which an ulcer had attacked the portal vein and led to a fatal pylephlebitis. Cases are described as pyopneumothorax subphrenicus in which an encapsulated abscess containing air had formed below the diaphragm. Osler has described a very unusual case in which rupture into the left heart occurred; nevertheless, the patient lived two days, for the perforation closed during every systole and opened during the diastole. Thus the patient gradually bled to death. Rupture into the pleural cavity may be diagnosticated when it leads to pneumothorax and empyema or to a direct communication with the lungs, and, as has happened, particles of food may actually be coughed up. In a case reported by Müller nematodes were found in the pleural cavity.

Rupture into the colon and subsequent lientery are rare. When, in a favorable case, the ulcer ruptures into the abdominal cavity, the preceding adhesive inflammation between the stomach and the neighboring intestinal wall and the omentum forms a cavity which is a sac within a sac, and prevents the propulsion of the stomach contents into the abdominal cavity. After this peritoneal irritative phenomena appear, with circumscribed pain, distention of the upper abdominal region, and fever, perhaps also severe vomiting. If the adhesions are very extensive, there may be complete stoppage of intestinal activity which, with permanent obstruction and increasing marasmus, leads to death, as in a case reported by Budd.

Perforation into the free peritoneal cavity is by far the most frequent complication, and happens either after preceding adhesion and abscess formation or without these. It may appear slowly and gradually or, on the contrary, the exit of the stomach contents may be slow. Adhesive abscesses then form which may later become encapsulated, or may rupture and produce general peritonitis. Usually perforation occurs suddenly without prodromes or, at least, without symptoms which point to such an accident. Without any cause or after a preceding trauma, such as an accidental blow, or pressure against the edge of a table or when leaning over a window-sill, in riding, after they have eaten heartily, or after or during vomiting, the patients suddenly feel severe pains in the abdomen which cause a feeling of collapse and, in a brief time, the picture of *perforative peritonitis* develops: Distention of the abdomen, obliteration of liver dulness, on even the slightest touch excruciating pain which may have a colicky or paroxysmal character, vomiting, singultus, facies hippocratica, small pulse, and with these symptoms the patient succumbs.

Spontaneous pain is usually referred to the gastric region; the ileo-cecal region is mentioned, that is, is sensitive to pressure, only in duodenal ulcers. According to the exhaustive compilation of Brunner, in a per cent. of all cases of perforation the history discloses the symp a preceding ulcer, which, upon the average, had been first noticed a years before. These may, however, be absent, and I remember a which a young girl, previously always healthy, collapsed at nigh dancing, succumbing to a perforating ulcer.

Such perforation may also be caused by spasmodic contraction stomach either from vomiting after medication, or from introduc finger into the pharynx, as is done by many patients to produce er and vomiting, or it may occur after introducing the stomach-tube. describes a case of perforation after vomiting induced by the The normal act of defecation is said by Bouillaud to have also cause.

The youngest female patient was nine years old, the oldest seve The youngest male was seventeen, the oldest seventy-two (Brunn

The seat of perforation in the gastric wall is usually the gr lesser curvature, very rarely the pylorus or the cardia. The lesio cular, with smooth, non-elevated borders, and surrounded by a less extensive zone of smooth, cicatricial tissue, which is usually so that at operation the suture will not hold but tears through, healing of the wound is exceedingly difficult.

Cases of such perforation that have recovered without opera among the greatest rarities. Brunner collected from literature the of 17, all of whom were treated with opium, ice, and rectal alim Almost invariably the perforation occurred a few hours after eatin the stomach was empty.

In these cases there is a justifiable doubt as to the correctne diagnosis. In a case described by Harland, as the author himself an ulcer with a simultaneously existing gastrectasis simulated per In this category I must also include the case reported by Spick spontaneous cure of perforative peritonitis in ulcer of the stoma was probably nothing but an acute gastric dilatation.

It is certain that the symptoms mentioned above may be a perforation may have occurred. I have seen two cases in wh was at first no marked distention of the abdomen, no shock or the pulse was regular, the temperature normal. It was obvious, that perforation had occurred. In both instances the stomach neither food nor gas, the patient for three days previously frained almost entirely from food, and, therefore, the rupture o was accompanied only by the signs of intense shock—loss of con Cheyne-Stokes respiration, absence of pulse, cold skin, etc.— abdomen was neither greatly distended nor very painful.

The "disappearance" of liver dulness, in particular, is a tain sign, as this may also be produced by the distended trans that extends high above the liver, or—in very rare cases—by a dilated stomach adherent to the liver.

Musser and Wharton calculate that perforation occurs in about 7 to 18 per cent. of all cases. If this includes the most marked cases only, even this low figure is, in my opinion, much too high. I have not observed more than 1.2 per cent. of perforations. Greenough and Joslin saw perforation in 3.2 per cent. of all cases. Those serious accidents in which several isolated ulcers in different areas of the stomach simultaneously rupture, usually in the anterior and posterior walls (Lovell-Keays and others), are very rare.

Occasionally a gastrocutaneous fistula forms with an opening into the epigastric or left hypochondriac region or between the ribs. This is a very rare occurrence; nevertheless, Murchison collected 12 reports of such cases. Sudden perforation has repeatedly awakened a suspicion of poisoning and led to unjust criminal trials.

The *nature of the cicatrization is of great importance.* It is obvious that cicatricial distortion may lead to the severest disturbance of the gastric functions, one of which, dilatation of the organ with cicatricial pyloric stenosis, has already been described; this produces a limited pathologic picture. In other cases, cicatricial contraction causes torsion of the nerves of the gastric wall or deformity of the organs, or the function of large portions of the muscularis is lost, or adhesions with neighboring organs form and lead to gastralgia or to functional disturbances which appear under the guise of "dyspepsia" of different kinds, the original cause of which is usually difficult to recognize and from which recovery without operation is, as a rule, impossible. In the course of years I have learned to dread this cicatrization even more than the primary ulcer. Not rarely such patients are erroneously considered as "nervous dyspeptics." If the cicatrix is circular and at about the middle of the stomach, the various forms of *hour-glass* stomach or large sac-like dilatations of the same are produced. In washing out the stomach the curious symptom then appears that the stomach apparently cannot be evacuated. After some time the water injected for washing returns clear, suddenly, however, again becoming turbid and admixed with gastric contents, and this phenomenon may be repeated several times. This indicates either the condition just described or an insufficiency of the pylorus, the contents of the duodenum being regurgitated into the stomach.

Hemorrhagic Erosions.—Einhorn has lately devoted much attention clinically to the previously mentioned "*hemorrhagic erosions,*" which have long been known anatomically, and frequently described. They do not present the classical picture of ulcer of the stomach but dyspeptic symptoms, even decided pain, which increases soon after taking food, and continues from one to two hours; emaciation and weakness appear. The chemism is not specially characteristic. Yet almost always in the water used for lavage—and authors lay particular stress upon this *constancy*—small reddish-white flocculi are present which, upon minute examination, prove to be desquamated portions of the mucous membrane. They show well retained glands with small cell infiltration and an accumulation of red blood-corpuscles between them.

These reports have been several times discussed (Pariser, Hemmeter, Platter, Ewald and others). That no positive diagnostic value can be attached to these mucous membrane particles which appear in the water after lavage, i. e., that they do not reveal histologic changes characteristic of a definite pathologic type, Leuk proved in my wards after thorough and careful investigation. His conclusions have been confirmed by Cohnheim, Lubarsch, Hari and others.

Elsner examined 35 cases in which inflammatory symptoms attributable to the stomach with a decrease or cessation of the gastric juice secretion were determined only 12 times, i. e., in 35 per cent., and found exfoliations of the mucous membrane either transitorily or permanently in the water used for lavage. Only four of these complained of difficulties such as Einhorn described. I have, however, elsewhere expressed my opinion (*Transactions of the Twentieth Congress for Internal Medicine,* Wiesbaden, 1902) that a *well characterized pathologic picture of hemorrhagic erosions does not exist,* and that every author who has attempted to portray this has presented a type differing from that of his predecessors. Not only the differential diagnosis of true ulcer, but also that between neuroses with hypersensitiveness of the gastric mucous membrane and spasmodic conditions of the pylorus, occasions perplexity. A positive diagnosis in such cases cannot be made from the course and the results of treatment; the methods of treatment which, under various authors, have resulted in a cure of "erosions," for example, lavage of the stomach with a one per cent. silver nitrate solution, will also cure an underlying gastric catarrh or the ulcer, which, as some observations invariably prove, may develop from such erosions. Henschen, in his autopsy reports, mentions erosions in 3 cases of tuberculosis, in 2 cases of nephritis (which I can corroborate, Ewald), twice in mental diseases, once in pneumonia and only once in gastritis, and these circumstances raise the question whether the erosions are not rather the consequence of a general affection than the expression of a local disease. In any case, the diagnosis "erosion" is very convenient, and will therefore probably maintain its position.

Fissures of the Mucous Membrane.—Here a change in the mucou membrane which is said to have its seat at the pylorus must be mentioned Fissures of the mucous membrane which produce pyloric spasm, just anal fissures give rise to rectal spasm. In his report upon the curati effect of large doses of olive oil in gastric disease (see under Treatment Cohnheim discusses these fissures extensively without positively demo strating their existence. He considers a fissure to be present from t fact of the rapid recovery, i. e., the cessation of pains, and maintains that the patients' symptoms could not be relieved in such a few days if they were due to well developed ulcer. In my opinion this argument does not warrant a positive diagnosis. For he has neither seen these hypothetical fissures himself, nor does he construct from them a characteristic symptom-complex, nor, finally—and this is certainly remarkable—are there any reports or proofs to justify this in the literature cited by him. In the many hundreds of autopsies in which I have examined the stomach with the closest attention, I have never seen such a fissure. Now, I will not deny that an ulcer may occasionally bear a certain resemblance to a fissure —a fissure is really nothing more than an ulcerated lesion of the mucous membrane—although, as the name implies, it must have more of a lacerated appearance than the ulcer which develops evenly—but it remains to be proven that these fissures are so frequent as Cohnheim believes, that among 24 cases (I exclude the 6 cases of undoubted carcinomatous ulcer) he made this diagnosis four times, therefore in 16.6 per cent. of the cases.

Syphilis and Ulcer.—In 1838 Andral propounded the question, Why do not syphilitic manifestations appear upon the mucous membrane of the stomach as well as upon the mucous membrane of the mouth? Since then this question has been much discussed, and more or less positive cases have been reported by Goldstein, Hiller, Virchow, Leudet, Lancereaux, Fauvel, Klebs, and Cornil. The simultaneous occurrence of gumma and ulcer of the stomach has been reported in only two cases. In others (Frerichs, Drozda, Murchison, Chvostek) cicatrices were found in the stomach and simultaneously general syphilis. Among 100 cases of ulcer, Engel found a preceding syphilis in 10 per cent., Lang in 20 per cent. Julien, in his great "Traité des maladies vénériennes," quite properly, is very conservative. In diseases so frequent as the two in question it must always be doubtful whether cause and effect or mere coincidence is before us, particularly as confusion with ulcerating gummata can by no means be always excluded. Only the result of specific treatment is decisive. Several such cases have been reported, for example, by Hiller and Gaillard, but the latter, who has written the most recent monograph upon this subject, admits that we have no certain proof. Specific symptoms are certainly not peculiar to syphilitic ulcers. Nevertheless, with coexisting syphilis and the signs of gastric ulcer it is advisable to institute specific treatment.

Tuberculosis and Ulcer.—Tuberculous ulcerations of the intestinal canal occur frequently, as is well known, but they are not often combined with ulceration of the stomach, perhaps for the reason that the dissolving gastric juice prevents the propagation of bacilli, whether introduced with swallowed sputum or with the blood. Typical symptoms are not peculiar to tuberculous ulcers of the stomach. Sudden death from hematemesis in consequence of eroded vessels has also been observed.

DIAGNOSIS

The diagnosis of chronic ulcer of the stomach is easy, a of the classical symptoms are present, can scarcely occasio When this is not the case it is exceedingly difficult or eve Important diagnostic factors have already been indicated; we fore, be brief. Two other diseases of the stomach, gastralgi dynia (as the expression of a functional nervous disturbanc cinoma resemble the symptom-picture of ulcer when ulcer d its typical course. It seems advisable to tabulate their imp of difference as follows:

Nervous Gastralgia.	Ulcer.	Ca
Tongue varies, often pale, and fissured at the borders or upon the surface.	Tongue dry, red, with white streaks in the center, or smooth and moist, or slightly coated.	Tongue pale, cases very
Frequent eructation of odorless gas.	Eructations either rare or acid eructations with pyrosis.	Frequent feti
Taste unaltered. Dryness of mouth frequent, sometimes salivation.	Taste unaltered.	Pappy, insipi
Appetite irregular, capricious.	Appetite good in the intervals. Thirst.	Appetite dec rexia. Ear to meat.
Varying sensations in the stomach, sometimes heat, sometimes cold.	Burning sensation in the stomach. Circumscribed boring pain, often radiating posteriorly.	Sensation of ing pains o acter, perha shoulder.
Spasmodic, burning pain, independent of food, often ameliorated by the latter or by pressure upon the stomach. Pressure points over the intestinal plexuses.	Pains gnawing, rare upon an empty stomach, usually appearing after eating or upon motion and on assuming positions which dilate the stomach; increased upon pressure. Pressure points upon back.	Continuous d of pain, p creasing to ten produc or increased
The chemism of digestion not especially altered.	Digestion of starches frequently slow; that of meat normal, or even accelerated; usually hyperchlorhydria.	Digestion ins ally absenc formation o ucts of deco

Nervous Gastralgia.	Ulcer.	Cancer.
Epigastric pulsation.	....	Epigastric pulsation only with marked emaciation.
Vomiting irregular, sometimes mucus only, sometimes more or less digested gastric contents, rarely admixed with bile.	Vomiting, as a rule, immediately or shortly after eating, and frequently the first symptom of the disease; very rarely without taking food, vomitus hyperacidas.	Severe and frequent vomiting, often periodical, occasionally also before the ingestion of food; mucoid; when acid, due to organic acids; only occurring during the course of other dyspeptic symptoms; vomitus shows but slight digestion; sometimes cancer cells present.
No hematemesis, except as accompaniment of very rare complications.	Vomiting of light red blood or coffee-ground masses; usually repeated in a brief space of time, occasionally very profuse, followed by extreme anemia and collapse. Compensation with comparative rapidity. Blood in the feces. Occult hemorrhages.	Decomposed blood more frequent than fresh; quantity usually slight, but, having once appeared, recurring frequently at short intervals.
Almost invariably stubborn constipation; normal evacuations very rare; occasionally fluid mucoid dejecta, the so-called pseudodiarrhea. Mucous colic, that is, colitis mucosa membranacea.	Bowel discharges vary, not infrequently diarrhea in consequence of intestinal irritation. Lientery after perforation into the colon.	Almost invariably bowels stubbornly constipated. Lientery after perforation of the colon.
No fever.	Mild fever only with adhesive inflammation after rupture of the ulcer, or following profuse hemorrhages.	Fever rare, and only toward the termination of life. Initial fever quite rare.
Skin pale, rarely ruddy. Skin of normal turgescence.	Skin usually of ruddy appearance, anemic only after profuse hemorrhages. Frequently the visible mucous membranes, and even the cheeks, slightly cyanotic. Patients sometimes present the chlorotic type.	Skin sallow, yellowish, dry, and flaccid. Marked cachexia.
Often conjoined with hysterical symptoms. Occurs at all ages, more frequently in women than in men.	Most frequent in middle life; rare in children. Accompanied by a varying psychical condition, frequently great depression.	Most frequent between the fortieth and sixieth years. Psychical condition that of depression; melancholia, but, strange to say, less profound than in severe cases of ulcer.

Nervous Gastralgia.	Ulcer.	Cancer.
No tumor on palpation, unless, as rare exceptions, when foreign bodies, hair, etc., have been swallowed. Chemism varies; absence of lactic acid.	When the ulcer is situated at the pylorus with consecutive hypertrophy, an ovoid, smooth tumor at the right of the median line may be palpated. Occasionally in old ulcers with a hard base and callous borders a palpable tumor with circumscribed encapsulation, perforation or adhesions with the head of the pancreas, the left lobe of the liver, the spleen or omentum, and does not move with the expiratory excursion. HCl present, and usually increased.	Tumor of varying size and shape, nodular or smooth, distinctly palpable: as a rule, passively moved, occasionally also in respiration. In the majority of cases no HCl; absence of pepsin digestion; lactic acid. Lab-ferment sometimes absent (cancer of the pylorus), sometimes present (cancer of the fundus). Secondary glandular enlargement. Metastases.
No symptom of perforation.	Perforation into neighboring organs with characteristic symptoms, frequently after apparent brief duration of the disease even occurring without prodromes.	Perforation; implication of neighboring organs only after prolonged existence of the disease.

Nevertheless, distinct as these three clinical pictures may appear to be upon paper, in practice the most prominent symptoms are often so ill-developed or so merged into one another that a precise diagnosis is impossible, as, for instance, in the onset of the ulcerative process. So long as the symptoms indicate only general digestive disturbances, so long as there are no typical gastralgic attacks, and especially so long as there is no trace of hematemesis, we have no clue by which to distinguish this condition from the great category of dyspepsias. An important aid in the recognition, which makes an early diagnosis possible, is the proof of hyperchlorhydria, although we should not forget that rare exceptions to this occur, and that chemically slight hemorrhages into the gastric and intestinal contents cannot be macroscopically recognized.

The diagnosis is most positive when we find the symptoms of typical gastralgia, vomiting of blood, blood in the feces, absence of tumor and cachexia. In such cases it is unnecessary to examine the patient with the stomach-tube; for this may be a serious procedure, and it had better be avoided. On the other hand, in indefinite cases the investigation of the chemism is absolutely necessary, but all precautions must be observed. The proof of hyperchlorhydria or of gastrosuccorrhea with a large amount of hydrochloric acid is then decisive, and indicates, particularly the latter occurrence, that the seat of the ulcer is at the pylorus, and there is consequent stenosis. I have seen patients with undoubted ulcer of the stomach with extreme loss of strength, and, on the other hand, cases of gastric cancer with unimpaired strength, appetite, and the general habitus. Occasionally, as Leube also states, the diagnosis can be made solely by the efficacy or fruitlessness of a specific treatment for ulcer. Extreme difficulty in the differentiation may be caused by the above described tumor-like cicatrization, which draws the neighboring organs toward the base of an ulcer to which they are adherent, or which forms above a perforated ulcer. In the latter case the head of the pancreas, the left lobe of the liver, or, more rarely, the spleen, may be involved. In the gastrocolic ligament there is a lymph-gland, or a band of closely situated glands, which under some circumstances enlarge and become sensitive to pressure; they may be palpated as small tumors the size of a hazelnut at the lower boundary of the stomach, and have repeatedly caused me the greatest perplexity in diagnosis. In all of these cases the persistent size of the tumor, the maintenance of strength, and the presence of hydrochloric acid, favor ulcer in opposition to cancer, just as a course lasting more than three years and the absence of a typical cancerous cachexia favor the first named affection. Since we know that hyperacidity exists in numerous cases of ulcer, it is obvious that those distinct tumors of the stomach, particularly those situated about the pylorus, which in spite of typical signs of malignant cachexia run their course with profuse excretion of hydrochloric acid, may be referred to the fact that cancer has developed upon the foundation of a gastric ulcer. I have repeatedly observed such cases. In several instances in which a tumor of the pylorus while under observation and within a year developed to the size of a walnut, the acidity amounted to 104 and 101. Gastroenterostomy was performed, and at this time the tumor was inspected and subsequently removed; it proved to be an unquestioned carcinoma.

A test of the chemism, as advised by Gluczinsky, is valuable for the early recognition of beginning carcinomatous degeneration of an ulcer and may be carried out upon one and the same day, first upon an empty stomach, secondiy, after a test breakfast, and, finally, after a midday meal. In a florid ulcer all tests give normal values; decrease or absence of one or more favors a beginning carcinoma. In 17 cases in my wards Dr. Sigel has put Gluczinsky's results to the test, and, although not in every case, in the main he confirmed them.

Diagnosis of Perforation.—The diagnosis of perforation of an ulcer is based upon the above described symptoms, and only those features will be discussed which may lead to error. Here we must consider perforation of a gastric carcinoma (a relatively rare occurrence), of the gall-bladder, that is, by a gall-stone, of the appendix, of an intestinal ulcer, and acute diffuse peritonitis caused by rupture of the spleen, of a pyosalpinx, or of an ovarian tumor. The greatest differentio-diagnostic perplexity may arise from the rupture of a gastric cancer, which, however, as has been mentioned, is extraordinarily rare because the neoplasm is attach itself by inflammatory adhesions to the surrounding organ other possibilities enumerated may be excluded by a careful exan and the history. But there are other diseases which, without produc foration and peritonitis, may simulate these conditions. Here a mentioned: Gall-stone and renal colics, torsion of pedicles, torsio ureters in movable kidney, severe gastralgia, poisoning, emboli thrombosis of the mesenteric arteries, and, finally, even pleur pneumonia, the latter affection occasionally, i. e., in a few case complicated by a similar symptom-complex. I can only call atte these occurrences; the diagnosis will be in part described in the ing pages.

Unfortunately, we have no means of recognizing a threatening tion because there is nothing to indicate whether, in a given are dealing with a superficial or deeply invading ulcer. Sympto sisting for a long time may, perhaps, be utilized in the latter se the uncertainty of such a conclusion is obvious, and the great of cases of ulcer run their course for years without perforation.

The diagnosis may be very obscure when we are deciding *cholelithiasis, renal colic,* and *gastralgia* occurring in the course of the pylorus or ulcer of the duodenum; of course, not in typi of either disease. Recurring pain in the right hypochondrium ind of the ingestion of food, slight fever, jaundice, enlargement of t pain over the liver, a palpable gall-bladder perhaps containin drawing pains along the ureter, hematuria, the passage of gall- renal stones are just as typical of gall-stone colic or renal coli total complex of the symptoms previously described is of ulcer. many cases the symptoms are so indefinite that confusion can sc prevented. If in gall-stone colic jaundice is frequently absent feebly developed, there are, on the other hand, not seldom case tralgia which run their course with mild jaundice, either becau forced into the blood-vessels from spasmodic contraction of the organs or because a rapid, transitory, sympathetic spasm of th duct occurs, and with this biliary stasis. The patients with colic are apt to locate their pain in the median line, particularl in whom the topography of the liver has been changed from laci even makes the very remarkable statement that among 403 cas stone colic which he observed in Carlsbad, in 380 = 94.3 per ce spasm occurred alone without pain in the hepatic region! If t is displaced somewhat to the right, or if the ulcer is situated i zontal axis of the duodenum, there can be no question of a local Hence, it may for a long time, or perhaps always, be a moote whether cholelithiasis or gastralgia is present. But here hyper of the gastric juice, if present, gives us a valuable clue. Acidity over 80, i.e., 0.3 per cent. HCl, may be utilized in this way.

A *tuberculous ulcer* is recognizable by its reaction to tuberculin, according to Petruschky's method. I should like to remark in this connection that we must never be content in such cases with one test if this be negative, but several injections, in increasing doses, must be given. Repeatedly I had no reaction with 1 and 2 mgm., but obtained a typical one after employing 3 and 5 mgm.

Position of the Tumor.—Only an unusually favorable combination of circumstances will enable us to recognize the position of an ulcer at the pylorus or in the duodenum, perhaps even in the greater curvature. On the other hand, by exclusion we may decide that the ulcer is situated elsewhere. An ulcer at the pylorus is characterized by sharp, localized pain a little to the right of the median line. Fleiner attaches great weight to the symptom of pyloric spasm. But the significant factor of pain cannot here be reckoned upon, and the assumption that ulcer situated in the cardia of the stomach is accompanied by sensations of pain immediately after eating while those at the pylorus produce pain only later has, I find, neither been sufficiently proven clinically, nor is it justified by the actual conditions. An attempt has been made to locate the seat of the ulcer from the position which some patients assume to alleviate their pain. If the pain is less in the left lateral position, the ulcer is said to be situated at the lesser curvature, or *vice versa.* This also is a very doubtful and uncertain symptom, inasmuch as it does not accord with the experience of the majority of patients. According to Gerhardt: "Sensitiveness to pressure and tumor favor the seat of the ulcer upon the anterior wall, pain in the back and hemorrhage its seat upon the posterior wall. The seat of the pain and its increase in the latter position often permit the differentiation of ulcer of the fundus or of the pyloric region. When an ulcer at the fundus is adherent to the spleen it may by producing splenitis give rise to chills, as I (Gerhardt) have seen in three cases." That gastrectasis indicates the seat of an ulcer at the pylorus or in the duodenum and contraction at the cardia, requires no special emphasis. When we reflect how vague is the symptom of pressure sensitiveness, how rare is the appearance of tumor due to ulcer in proportion to the total number of cases, how very difficult is it in these cases to determine the constriction of the stomach *intra vitam* since we do not inflate the organ or introduce the stomach-tube, and, lastly, when we remember that often several distinct ulcers are situated in different areas, no great weight will be attached to this symptom.

But, in my experience, the most reliable of the symptoms which have been mentioned is the pain, often spasmodic, in the region of the pylorus, therefore, in the right mammary line, localized below the border of the liver. As, however, the pylorus is occasionally very movable, and may frequently be found even at the left of the median line, it is c pain in the latter region cannot always be referred to the great ture or even to the fundus. v. Leube very correctly remarks: " beware of such diagnoses. They are at least uncertain, as we diagnoses based upon the subjective symptom, pain, even when s cial condition is pointed out by this; for instance, when pain is (only on displacing the gastric contents from a particular positio fore only appearing when the patient assumes the right lateral] Nor must we forget the well known fact, to which Schütz has again called attention, that there are inflammatory conditions in t verse colon associated with pain which may readily but errone referred to the stomach.

It would materially aid us in the comprehension of this con we possessed a really good *gastroscope,* but, at present, this does n to be the case. It need hardly be stated that the same considerati deters us from introducing a stomach-tube or a soft sound in more strongly deterrent in the case of a rigid metal tube.

It is true that a stenotic gastric ulcer at the pylorus with co gastric dilatation, and, *vice versa,* the same condition at the car resultant contraction of the stomach, are readily recognized, th perhaps, requiring the aid of the esophagoscope; but, even with get little further knowledge, and to the cases mentioned by Ge may oppose another where the perforation of a broken-down c of the smaller curvature manifested itself by chills and intense p cially upon the left side. We are in an extremely difficult posit several ulcers are simultaneously present, and this condition is *Exact knowledge of the location is, therefore, for many and pe most cases, very desirable,* and the more important because accu tion of the seat of the ulcer is of the utmost significance in the t and particularly for operative intervention when hemorrhage occ difficult this determination is may be understood from the Schloffer was unable to find it in 2 of 5 cases even at the opera

But even if we were so fortunate as almost to grasp the our hands, i. e., to detect *tumor at the pylorus,* and although oth particularly the chemism of the stomach, the age of the p general condition and strength, were of such a nature as to e cinoma, an absolute diagnosis could not be made. Hence the foll ditions come into view, and these we must discuss for a few m

I. *Pylorospasm.*

II. *Muscular hypertrophy,* that is, *cicatricial thickening of region.*

III. *Carcinomatous neoplasm.*

Of the first it must be remarked that although its most fre is an ulcer at or in the immediate vicinity of the pylorus, case undoubtedly observed in which pylorospasm occurs without any alteration in the mucous membrane that can be detected. Here the observations of surgeons are of great weight. Both Schloffer and Alberti have reported such cases.

The case of the first author was that of a woman, aged 44, in whom the gastric contents showed no hydrochloric acid, but lactic acid. At the operation an oval, slightly diagonal, extraordinarily hard, circumscribed resistance of about 2 cm. in length and 1 cm. in breadth, which prolonged investigation showed to be nowise altered, was found at the greatly narrowed pylorus, and on its posterior wall. The area in question was resected, and, most astonishing to relate, absolutely no pathological changes were found. Histological examination merely showed a slight hypertrophy of the musculature in this area, and Schloffer assumes the case to be one of circumscribed spasm of the musculature of the pylorus in its posterior wall.

The case of Alberti is interesting from the fact that at the laparotomy a pyloric tumor was found which was of uniform hardness, sharply demarcated toward the duodenum, but toward the stomach and particularly at its posterior wall it showed merely a coarse and firm wedge-shaped swelling amounting to about 4 cm. in length. Alberti looked upon it as a neoplasm, and concluded to perform pylorectomy, the more so since no adhesions or metastases were apparent. When, however, the lesser and greater omentum were detached, all signs of tumor suddenly disappeared, and the stomach, that is, the pylorus, became soft and perfectly normal. Pyloroplasty after Heinecke-Mikulicz was performed, and the pylorus was found to be narrowed but no firm cicatrix could be detected. The attacks of intense pain were arrested, and the patient, in spite of the continuance of symptoms of stenosis of the pylorus for five years, showed very slight dilatation of the stomach.

This case appears to leave no room for doubt. Here marked reaction for hydrochloric acid was always present. On the other hand, in the light of our experience in the Augusta Hospital, the case of Schloffer may be regarded as questionable.

We have observed two such cases in which resection was apparently performed on account of a benign pyloric tumor, and the most minute histologic examination of the resected tissue not only by us but also by our pathologists showed no change characteristic of carcinoma. The first case was that of a woman, aged 47, who had been suffering for several months from a gastric affection, and complaining of vomiting and eructation; examination showed a moderate gastric dilatation and a pyloric tumor causing stenosis. Free HCl was repeatedly found to amount to 0.5–0.6 per cent., albumin digestion to about 75 per cent. Lactic acid and blood were never present. The gastric tumor was resected and gastroenterostomy performed. In 20 sections from the resected tissue nothing characteristic of cancer could be found. For five years the woman remained in good condition, then ascites developed combined with abdominal symptoms which pointed to a tumor of the abdomen. An indistinct resistance could be palpated both by the vagina and by the rectum.

Operation performed in the summer of 1901 revealed extrauterine pregnancy and, simultaneously, a neoplasm of both ovaries which, at the histologic examination—the patient died soon after the operation—proved to be an adenocarcinoma. There can be no doubt that this was a metastasis from the stomach.

The other case occurred in a man, aged 37, who, six weeks prior to Christmas of 1897, was suddenly attacked by diarrhea which continued until the time of his admission upon the 9th of March, 1898. Fourteen days previously he began to vomit brownish masses, but had never suffered from pain in the stomach; he felt quite strong, and had a good appetite. After inflation of the stomach a tumor the size of a small apple was found in the epigastric region upon the right below the border of the liver. The stomach was slightly enlarged; no glandular enlargement. Acidity was 58 upon the empty stomach and lactic acid 25. In the test breakfast, acidity was 54, lactic acid 16 (0.058 per cent.). Pepsin digestion was barely 10 per cent. Laparotomy upon the 14th of May disclosed a tumor of the greater curvature extending to the pylorus, and in its middle a deep ulcer. About two-thirds of the stomach was resected, and on the remainder of the stomach gastroenterostomy antecol. anter. was performed. The patient was discharged cured upon the 7th of April.

Although the diagnosis of *carcinomatous ulcer* was made from the macroscopic appearances, histologic examination did not reveal a characteristic picture. The man has just been readmitted to my ward with a large, undoubtedly malignant tumor of the greater curvature.

We see, therefore, that in the differentiation of these so-called spastic tumors and muscular hypertrophies of the pylorus we must be very cautious. It is not surprising that, in spite of resection of an apparently healthy organ, metastases should develop. Years ago I stated that typical, cancerous nests might be found in the submucosa and muscularis far beyond the macroscopic, visible portions of a malignant tumor.

Tumors due to muscular hypertrophy are generally differentiated from spasm by their consistence. The same cause which generates them always produces stenosis of the pylorus, whether they originate from the cicatrix of an ulcer causing stricture, or from reflex contraction, from the irritation of a fresh ulcer by the ingesta, or, finally, from a malignant neoplasm. I must also mention that occasionally a gall-stone is incarcerated in the pylorus (Naunyn, Elsner) or tumors from hair or trichobezoar may be present. In how far dilatations of the stomach occur in connection with these conditions, and how far they may be attributed to it, depends upon the duration of the affection, upon the permeability of the stenosis, and upon the degree of the compensatory muscular hypertrophy of the stomach. Here, however, there are marked gradations in the condition. We meet with convincing proof of the fact that ulcer of the pylorus frequently merges into carcinoma of the pylorus and here, as in other areas, persistent mechanical irritation from the chyle which presses through the pylorus performs an active part. These are the cases in which hydrochloric acid secretion and the peptic function of the stomach are long retained, although they finally decrease from the norm. With these also belong some of the cases which I first mentioned, cases in which, on account of the chemism of the stomach, the diagnosis of a benign tumor of the pylorus was made, while operation or autopsy revealed that we were dealing with a more or less carcinomatously degenerated old ulcer. It always appears to me that the general condition of such patients suffers but little provided high-graded pyloric stenosis does not occur, and this is another factor which may lead to diagnostic errors.

In conclusion we must consider the last of the three groups mentioned, namely, *carcinomatous tumors situated at the pylorus or in other portions of the wall of the stomach.* Here, of course, we are only dealing with those cases in which the age, the patient's strength, the chemism, the gastric contents, and the objective condition raise doubts as to whether we have before us a carcinoma, the old cicatrix of an ulcer, or an epigastric process.

In my opinion, during the short or long course of the disease a certain grouping of the symptoms may sometimes entirely preclude our making a diagnosis at the time, because neither the individual symptoms nor their totality are sufficiently clear to lead in any direction to a decision. This is particularly true of two symptoms to which we otherwise, quite justly, attach great significance: The *pain* and the *emaciation.*

Here I entirely leave out of consideration those cases in which gastralgia occurs at the initial stage of pulmonary phthisis in young, chlorotic and anemic persons or where these attacks occur in the so-called preataxic stage of tabes dorsalis (locomotor ataxia), because these may be easily differentiated by a minute examination which, in women, should include also the genitalia.

A number of years ago Aufrecht called attention to cases of severe gastralgia without anatomical findings which even showed a family relationship. I have never seen cases of this kind, and must say that Aufrecht's brief report does not appear to me convincing: "At the autopsy nothing was found in the stomach, the biliary passages, or the duodenum to make clear the cause of the disease."

In old gastric ulcers the *typical pain* beginning at a definite time after the ingestion of food is either absent, or it is so irregular that it does not differ from cardialgia due to other causes; therefore, it does not differ from the pain produced by perigastric adhesions and carcinoma, either typically, or by its seat, or by the frequency of its appearance. we must also consider the typical pains due to a *hernia of the linea* In general these may be readily demonstrated, but it is necessary to them in mind, for, in my experience, they lie somewhat beyond the of the experience of most practitioners. By proving that the con was hernia, and by a slight operation, I have succeeded in pro curing many a case diagnosticated as gastric ulcer.

If, however, there are no well developed attacks of pain, and w dealing only with more or less decided discomfort, a sense of pre and fulness after eating, we know that these may exist for a long and, even until death, be the only symptoms in patients with cance

It is true that in ulcer emaciation is usually much less marked th cancer, and that the color of the skin characteristic of malignant ca is usually absent. Where, however, an ulcer forms in debilitated, ne hysterical persons or, *vice versa,* in the robust who, from a fear of limit for a long time the amount of their food, rapid and striking of weight occasionally occur and lead us to consider seriously the ence of carcinoma.

But the condition of the peripheral lymph-glands should give us ble information on this point!

Long ago, in 1886, Dietrich proved, after a careful examinat normal persons, that is, those not suffering from malignant disea great frequency of slight glandular enlargement—thus, for examp inguinal glands in 92 per cent. and the axillary glands in 64.9 pe were swollen to the size of a bean. But decided tumefaction m present for this to be of any importance. Tarchetti lays special upon enlargement of the supraclavicular glands which he met w 18.4 per cent. of cases of cancer. But in my experience statistics of g lar enlargements *are not very reliable.* In 125 cases of positive u which I have notes regarding this, sixty times the inguinal glan enlarged to the size of a bean, and twenty-four times the glands axillary cavity to the size of a pea. Nevertheless, I must admit t condition of the lymph-glands, particularly the enlargement of th sided supraclavicular glands, is of some significance. Still greater must be attached to the state of the *tongue,* which in ulcer—espe recent ulcer—is red, moist, glistening, while in cancer it has coating and is usually dry, in nervous disturbances it is deeply f particularly upon the sides, so that the tongue resembles a freshly p field, or, to use a more striking comparison, a glacier with its cleft this, fortunately, again brings us to the symptom *per tot discrimin* to which the ancients, without any knowledge of our modern n attached so high a diagnostic value!

Duodenal Ulcer.—What is true of the seat of ulcer of the sto also true of the seat of ulcer of the duodenum: in at least 90 p of the cases it is impossible to decide positively whether we are dealing with ulcer of the duodenum or ulcer of the stomach. The duodenum, and particularly its horizontal branch, so far as these conditions are involved, is only to be regarded as an appendage to, or a portion of, the stomach, and ulcerative processes in one, as in the other, produce the same symptoms. Factors which favor ulcer of the pylorus are also operative for ulcer of the duodenum, all the more so as the ulcer sometimes directly attacks the duodenum from the pylorus. A *duodenal ulcer* is likely when the pains only set in some time after the ingestion of food; their seat, as well as a passive sensitiveness to pressure, is decidedly to the right of the parasternal line, and profuse hemorrhagic dejecta and hematemesis may appear. Jaundice and peritonitis have been repeatedly observed. The fact that ulcer of the duodenum frequently occurs in old persons after extensive cutaneous burns may in a given case be of value in the diagnosis. A point of support, but no more than this, is the rare occurrence of duodenal ulcer, for, according to Willigk, to 225 cases of gastric ulcer there are only 6 ulcers of the duodenum, according to Trier only 28 to 261. But this small percentage includes a number of cases in which ulcer was present in the stomach and duodenum simultaneously. Gastralgia is said to be not so frequent as Budd believes, because the duodenum is exposed to less distention and less change of position than the stomach. Jaundice, which is very rare in ulcer of the duodenum, can be little utilized in the diagnosis from the circumstance that, in the main, intestinal hemorrhage is more frequent and hematemesis rarer, but gastric ulcer also leads to hemorrhage from the intestines, and ulcer of the duodenum may be accompanied by hematemesis.

PROGNOSIS

Until recently, and quite properly, a doubtful prognosis was given in gastric ulcer when it was differentiated by the signs which have been mentioned. Since we have become able to make an early diagnosis, and to separate it from the forms of dyspepsia, since the principles of treatment have been clearly established, and we are in a position to use them at the onset of the process, the prognosis in at least the early stage of ulcer has decidedly improved. If the patient is subjected to rational treatment at the proper time, i. e., if a rest cure is instituted, well founded hopes of recovery may be entertained, and even in classical ulcer recovery or decided improvement may be looked for. Unfortunately, during the first stages, which subjectively do not occasion great difficulty, very few patients are inclined, or are in a position, to submit to such treatment. If, however, we succeed in curing the ulcer by permanently changing the abnormal composition of the blood or the secretion of the gastric juice, the fear of relapse is also removed, which, otherwise, always threatens and only too frequently occurs. Invariably, however, and particularly i the healing of extensive ulcers, there is danger of a permanent damage t the health from the consequences of cicatricial tissue formation, and th cannot be lost sight of. In such cases the prognosis must be made wi great caution. That it is not bad is, nevertheless, evident from the we known fact that cicatrices from gastric ulcers are found about twice frequently as open ulcers. In hemorrhage, provided it is not immediate fatal, the prognosis is, as a rule, favorable, and it is better the young the individuals in question. By suitable treatment we usually succeed mastering the hemorrhage, and even extreme anemias are improved in relatively short time. At the Surgical Congress in Berlin (1897), Leube gave the following statistics of his cases: Of 424 hospital patie 314, = 74 per cent., were cured after a treatment of four weeks, 93, = per cent., were improved, 10, = 2.4 per cent., died, 7, = 1.6 per ce were unimproved. Therefore, in only 4 per cent. was careful treatm without result. This coincides with my reports of 233 hospital patie in which I had 76 per cent. of recoveries.

But it cannot be denied that in these statistics the factors, which l often been mentioned, of unlike circumstances and insufficiently long servation play a part. The surgeon reaches other results than the pl cian, and thus it comes to pass that v. Mikulicz gives a mortality of 1 20 to 30 per cent., while Leube has only 4.1 per cent. It is obvious the brief period of observation in the hospital can only be regarde corresponding to the "healing" of the ulcer; it does not strictly a to the period after the patient's discharge from the hospital, and us nothing of the mortality. The latter depends upon accidental ditions, and can only be arrived at with some degree of certainty the reports of many autopsies. But the "statistics of cures" are open to doubt from the fact that groups denominated as ulcer in many cases in which true ulcer was not present, as is evident fror nature of these cases. For this reason I have refused to utilize my s tics in this way. J. Schulz has decidedly cleared the situation. analyzed 291 cases, 184 from the Breslau Clinic, and 107 from the E dorf Hospital, all of which were recognized as ulcer by the appe of hemorrhage. Of course under these circumstances a diagnostic was not absolutely excluded, but, nevertheless, it was scarcely po Questions were sent to all of these patients from which deduction to be made regarding the results of treatment. One hundred and seven answers were received, and the accurate investigation of these fied the conclusion that internal treatment of ulcer of the stomach ing to the method instituted by v. Ziemssen and Leube gives a nently good result in 64 per cent. In 18 per cent. relapses occurre nevertheless, most of these patients were finally cured. It might sumed that under renewed proper treatment the others would de improve. In 18 per cent. the treatment was without result, and of these 7.6 per cent. died. If, therefore, we count those who were not benefited among the unsuccessful cases, 64 per cent. were cured, in 13 per cent. there was temporarily either no result or a relapse, and in 23 per cent. absolute failure.

TREATMENT

Whenever possible, gastric ulcer should be treated by the rest cure inaugurated by v. Ziemssen and v. Leube, in which all irritation of the stomach is to be prevented, just as a fractured bone is immobilized by a plaster dressing, naturally with the difference that in the latter case this rest is absolute while in the former it is only approximate. Rest in bed and nutrition by the rectum or by food which burdens the stomach as little as possible are the foundations of this treatment which, in England, was long practised by Wilson Fox and Balthazar Forster. As adjuvant, anodyne remedies, and at the same time calculated to lessen irritation, moist heat in the form of hot compresses and the drinking of hot Carlsbad water or a solution of Carlsbad salt which neutralizes acid, are recommended. v. Leube administers the Carlsbad water lukewarm; the compresses, however, should be as hot as they can be borne; according to his latest reports, he no longer uses nutritive enemata. To the latter, however, I attach the greatest importance, while I have discarded the hot compresses, chiefly because they leave an ugly pigmentation upon the abdomen. It is astonishing how well the majority of patients will bear exclusive rectal alimentation for three, four or more days, the nutritive property of which, according to all recent investigations (Eichhorst, Ewald, Huber, Rost and others), and in spite of several attempts to depreciate its practical value (Plantenga, v. Mering), admits of no question, particularly if small quantities of a 5 per cent. solution of cocain upon pellets of ice be given by the mouth.

Whether nutritive enemata, as Zierko maintains, actually diminish the acidity of the gastric juice, has not yet been positively determined.

Acting upon Kussmaul's advice, I have for some time administered large doses of bismuth in suspension (about 15 to 20 grams in 200 of water daily, divided into three doses, well shaken, and given before meals) which, according to the experimental reports of Matthes, forms a protective coat over the exposed, ulcerated surface.

This treatment, according to universal experience, gives excellent results where the ulcers do not invade too deeply, or where the condition resembles that of a florid ulcer, while, in truth, other affections—perigastric adhesions, neuroses, cholelithiasis, renal stones and the like—are the causative agents. Bourget has lately declared that rectal alimentation is rather harmful than beneficial (why?); on the other hand, he administers rice soups and milk and rice, and, therapeutically, washes the stomach with a 2 per cent. solution of iron chlorid, to which ½ per cent. of potassiu chlorate is added; so far, I have had no occasion to test his method.

Leube attaches weight to the fact that Carlsbad salt has a neutralizin and, on account of the sodium chlorid it contains, also a stimulatin effect; but the latter might rather be looked upon as deleterious, sinc we know that in many cases the acidity is greatly increased, and ther fore a depressant rather than a stimulant is indicated. As to the neutra ization, or decrease, of the acidity, I do not attach much importance this if it is observed but a single time in an empty stomach, provid there is not always a secretion in the stomach when it should be empt On the contrary, the diminution of the hypersecretion and the laxati effect of the neutral salts, as well as the soothing influence of large qua tities of warm water, appear to me to be of the utmost importance. the water of simple alkaline springs has been found to be less effecti than that of the saline alkaline springs, it is only because we have f gotten to produce the desired laxative effect by other means.

Where there is no laxative effect from Carlsbad water, as is frequen the case, it must be brought about by the addition of Glauber salt still better, by vegetable laxatives, preferably rhubarb or senna. It unnecessary to adhere rigidly to a formula, the principle alone is imp tant. Whether the pains are relieved by warm fomentations or, in these are ineffectual, by small doses or subcutaneous injections of phin, whether the patient is given a solution of Sprudel salt or the nat spring water from Carlsbad, or, for example, Ems, Vichy, or the N nahr Spring, and the absent neutral salt be supplied by the addition other aperients, is immaterial. Of the Carlsbad spring water 300 to c.c. should be given. But which spring? This is unessential since t are no important differences in the chemical combination, and the di ences in temperature of the individual Carlsbad springs are of little sequence because these waters can only be taken as hot as the patient bear them; therefore are all taken quite hot. Fifteen grams (about tablespoonful) of the salts should be taken daily, small quantities solved in half a liter of water at intervals, with corresponding p between. For the first three days absolutely no food is to be adm tered by mouth, but a nutritive enema is given three times daily; s quently, besides the enemata, milk, or milk in flour soup, in tablespo doses, or bland pigeon or chicken broth. The milk, on account o fine floccular coagulation, has some pegnin added. If this diet is borne, it is added to in a manner soon to be described; otherwise, ab rectal nutrition is again instituted. If no pain follows the careful a istration of milk, we may permit somewhat larger quantities (up flat plateful, i. e., about 180 c.c.), leguminous flour soup, then leg later pappy food made of chestnuts, sago, tapioca, Kufeke's flour, hy and others, and later small quantities of meat. Among nutritive [illegible] milk takes the first place. It was first advised for this purpose by [illegible]. It is suitable because it contains all of the nutritive elements in solution, that is, finely divided, is free from irritating substances, because the acid is neutralized and because the casein which forms from the action of the gastric juice remains soft. The patient, however, must drink it very slowly and carefully. To prevent the fermentative coagulation of the milk and the irritation of the intestinal surfaces due to this, I now add [illegible] which produces a very fine flocculent coagulation. [illegible] for patients who cannot take milk. [illegible] the white of an egg and [illegible] may be given. We must limit ourselves to these foods until the severe symptoms have disappeared. In the third week a food richer than the [illegible] and [illegible] is permissible, and we should then [illegible] food of somewhat greater consistence, such as [illegible] eggs, [illegible] of the breast of [illegible] but milk is always preferable, and we should always be ready to return to a simpler diet as soon as [illegible] pains appear.

[illegible]

[illegible]

[illegible]

[illegible]

The pains ceased almost immediately after the ingestion of food rich in albumin, and the patients recovered more rapidly than those on the preceding diet. Of 25 patients who were subsequently questioned, 18 (72 per cent.) were entirely free from symptoms.

The editor of the *Fortschritte der Medicin* quite properly remarks of these reports that the results are no better than with the old, reliable diet, and that there are very few who would risk the employment of such food in the stomach of a patient with ulcer. The reason given by Lenhartz-Wagner in favor of this coarse diet, that the patients recover more quickly, does not appear to me to be justified. Patients with ulcer recover very rapidly when the pains cease, and this takes place the more quickly when the gastric mucous membrane is allowed absolute rest instead of being stimulated to greater activity.

It is true that patients on this diet are at first debilitated and lose weight, but even here we must individualize. To attempt to regulate the diet in these cases according to calories is a form of play under a scientific cloak which to-day is much in vogue. As soon as the patients are able to eat at all they are prone to eat so much, even without calory force, that they soon recover from the former undernutrition. Convalescence is rapid, gastralgia ceases, and the time arrives when we must consider the second indication, the strengthening of the constitution.

For this purpose *iron preparations,* either alone or in combination with arsenic, are serviceable, the former in cases of pure chlorosis or anemia, the latter when we are combating a weakened nervous system, and it is desirable indirectly to influence this by the direct stimulation of metabolism. The old opposition to the employment of iron in ulcer of the stomach was founded on the experience that iron is frequently badly borne while the florid process is present; this, however, does not hold good when improvement sets in. Which iron preparation is to be employed depends largely upon personal preference; each day brings forth a new one, and in one case this, in another that, is the better tolerated. Of late, I have frequently employed triferrin which, without exception, was well borne. I formerly used arsenic in the form of Fowler's solution with tinctura ferri chloridi. After Liebreich's investigations arsenious acid appeared to be more serviceable and I now employ this in pill form, 1.5–2 mgm. of arsenious acid, 2 cgm. of ferri sesquichlorat. Considerably smaller but decidedly active doses of iron and arsenic, as has been proven by investigations in metabolism conducted by Ewald and Dronke, are given in the waters of Levico and Roncegno and the Guber Spring, which are excellently borne. The remedy should be given in increasing doses and after meals. This régime must be continued for months, and then the administration of arsenic must be interrupted for three to five days about every three weeks. The diet may gradually become more liberal but, nevertheless, must be strictly regulated for months, and patients who show a tendency to exceed their allowance must be given a written diet list indicating the amount of food permitted.

It is sometimes impossible to carry out the rest cure mentioned above because there are many patients who are unwilling, or not in a position, to undertake it, and in many cases it is necessary to fulfil a stringent *indicatio symptomatica.*

Under these circumstances treatment with large doses of bismuth is recommended. Kussmaul (Fleiner) advised that this in suspension be introduced in large doses into the stomach previously washed out, and that the patient then, according to the suspected seat of the ulcer, assume for some minutes such a position as will enable the drug to sink to the lowest area; therefore, for example, in ulcer of the pylorus the right lateral position. Since, as a rule, I avoid lavage in ulcer, and because I know that the stomach is usually empty early in the morning, and also that by a previous drinking of water the gastric juice present may be diluted and more rapidly propelled into the intestine, and since, moreover, it is scarcely possible so to cleanse the gastric mucous membrane as to remove particles which may possibly be adherent to the ulcerated surface, and this, at all events, would necessitate the use of many liters of water, therefore I omit the washing and permit the patient to drink the bismuth suspension upon an empty stomach. The beneficial results I have obtained prove the reliability of this modified process. Indeed, the patient's tolerance of the drug and the ensuing freedom from pain are quite remarkable, although not so invariable nor so prompt as would appear from Fleiner's report. This is quite natural; the conditions are occasionally much more complicated than we assume and the method presupposes to be the case.

The dose is large, apparently unlimited, and may be given for an indefinitely long period. In one of my cases the patient received over 800 grams in the course of a few weeks without the slightest inconvenience, not even constipation resulting. In this case, however, considering the size of the dose, the result was not very satisfactory. The same may probably be inferred from the large quantities employed by Fleiner (up to 1,000 grams). In place of bismuth, Pariser advises equal parts of the cheaper white chalk and talcum, one to one and a half teaspoonfuls in water in the morning upon an empty stomach, and in the evening three hours after the full evening meal. Under this treatment the feces remain light in color, and small hemorrhages are much more readily recognizable than in the dark bismuth feces. In place of bismuth subnitrate, bismuth subcarbonate (Boas) and bismutose have been advised.

If all proceeds as we anticipate, and a protective covering of bismuth forms over the ulcer, this has not only a symptomatic but a curative effect. Under this protection the ulcer has time to heal, and, as Matthes has shown, the formation of granulation tissue at the base of the ulcer and the proliferation of glandular and other epithelia take place.

Under thorough treatment which is long enough continued, in perience for at least four weeks, recent ulcers promptly heal. I cases a single treatment is not sufficient, but a repetition is ne The result of this treatment, however, is generally so uncertain th an unsuccessful issue we may at once assume either that an i diagnosis has been made (particularly the pure neuroses lead to or that we have old, deeply invading ulcers with broken down, ex borders, that malignant degeneration has begun or induration ha oped. In such cases other curative measures are indicated,—gastric tonics, and stomachics, small doses of mild narcotics, aperients, like. The indications for the employment of these remedies n obtained by testing the functional activity of the stomach.

Of additional remedies silver nitrate has also been advised, solution, and in decided doses (0.2 silver nitrate to 200.0 of water, spoonful every two hours). With this remedy I have sometim decided amelioration of the symptoms, and even a complete cure; i instances, after a little time the remedy had to be stopped beca creased disturbance of the stomach, nausea, anorexia, coated tong derangement of the intestinal activity, diarrhea or constipation, ap In one of my cases, every time a spoonful of the silver solution wa watery dejecta accompanied by severe abdominal pain followed.

In my opinion the dietetic principles enunciated above are a portant in the treatment of ambulatory cases, and the diet should, be followed as far as possible. As the patients digest meat bett starches and vegetables, they instinctively eat less of the latter, an ually, therefore, suffer from a monotonous meat nutrition, i. e., th ciate, and become nervous and irritable. This must be counter much as possible, and larger quantities of fat in the nourish not contraindicated because, as I showed some years ago, fat has t of decreasing acidity. I endeavor to modify the hyperacid gast by repeated small doses of alkali combined with rhubarb and ca or milk sugar. Rhubarb has a mild action upon the intestines, t has a decided anodyne effect, and for this purpose has been re advised. I use a powder of about the following composition:

℞ Magnes. ust.,	āā	5
Natr. carbon.,		
Kalii carbonic.,		
Pulv. rad. Rhei		1
Sacch. lactis		2

M. D. S.: Every hour enough to cover the tip of a knife.

This is to be taken dry; I have seen good results from its use. who know by experience the beneficial effects of alkalies, parti soda, are usually afraid of taking too much. In this respect their minds may be easy. I have never yet observed a deleterious effect from the too long-continued use of an alkali, particularly of sodium bicarbonate.

For the catarrh which accompanies ulcer, Ord advises potassium iodid with the addition of sodium bicarbonate in about the following formula:

℞ Kalii iodat.................................... 2.0
Natrii bicarbonic.............................. 5.0
Acid. hydrocyan. dil. (m. 2 per cent. acid) gutt. tres
Inf. rad. Gentian..........................3.0: 150.0
M. D. S.: A tablespoonful three times a day.

It is well to remember the advice of Pariser that women who have suffered from ulcer, even after treatment is discontinued, should be kept in bed during the next two or three menstrual periods and on almost the same diet as while suffering from ulcer.

For the severe gastralgia, morphin internally or subcutaneously ranks first. Chloroform solutions (1–120, a tablespoonful every two hours) or chloroform in drops (5 to 6 in a teaspoonful of water or on a pellet of ice) occasionally produce excellent effect, not only upon the momentary pain but upon the course of the process in general. For a long time I have been in the habit of administering bismuth in suspension with a one per cent. addition of chloroform, or, instead of using distilled water, I employ chloroform water. Tincture of iodin, 5 drops, three times daily in water, occasionally acts as an anodyne. Of other sedatives I have occasionally employed lupulin, extract of cannabis indica, extract of hyoscyamus, and extract of belladonna, but have always been compelled to return to morphin or codein. Cannabis indica, in particular, which is so greatly praised and was advised by G. Sée, has in my hands repeatedly failed to show any anodyne or quieting effect, but, on the contrary, has produced an unpleasant irritation. Formerly leeches were applied at the seat of the affection, and also blistering plaster; it was painted with strong solutions of iodin, and even the actual cautery was used. We are content to-day with the ice bag, or the application of ice-cold or warm compresses, or Leiter's coil which, where the circumstances permit, is the most cleanly and convenient mode of employing cold.

Treatment with Oil.—Treatment with *oil* (linseed oil or olive oil) has been praised by various authors. Cohnheim, in 1889 and later, was the first to report surprisingly good results. The oil is to be given in gradually increasing doses from a tablespoonful to a wineglassful or may be poured in through a stomach-tube. Agéron advises the admixture of 10 grams of dermatol with 200 of the finest linseed oil or poppy seed oil. Walko adds bismutose or bismuth. The oil is best taken while the patient is in the recumbent posture, and Agéron has the patient assume the dorsal decubitus, raising the pelvis after taking the remedy to relieve the gr curvature. Aside from the fact that the stomach-tube should not be i duced in cases of ulcer without very clear indications—which I thin not include the pouring in of oil—I have previously cited a case in v the oil caused such great nausea and retching that it was the imme cause of a severe gastric hemorrhage. Cohnheim, however, in abo cases, even with irreparable organic changes at the pylorus as well functional disturbances, has seen no unpleasant secondary effects but ally a very favorable influence upon the pain, the spasm, the g nutritive condition and the degree of acidity of the stomach. Even in which operative interference had been advised by others were by this treatment. According to their nature, cases of the first ca can only be symptomatically influenced, while permanent results actually been attained in spastic stenosis of the pylorus, in ulcer, sures (?), in erosions of the pylorus, in pyrosis hydrochlorica wit gastritis, and similar conditions when a pure neurosis formed the f tion of the difficulties, and provided no complicating perigastric pr existed. Instead of oil, an emulsion of almond milk (about a tablesp of sweet almonds ground up in about 200 of water) also has a q but not a nutritive, effect. The oil has the following merits: It spasm, it lessens friction, it decreases the acid secretion, and it in the nutrition.

Cohnheim, in his last publication (1904), remarks that it is that his reports have been confirmed only by Walko. This may two reasons. Either the process has been generally satisfactory, a by accident others have failed to praise it, or the results lauded b heim have not been attained by others, and judgment has been sus The latter describes my own position. Ever since Cohnheim's fi lication I have repeatedly employed the oil treatment in my priva tice, in the Hospital, and in the Clinic, and I have observed some b and also some very ill, effects. The oil was so repugnant to man patients that it was impossible for them to take it, and in others quently caused complete loss of appetite, eructations and vomi matter whether given by the mouth or introduced through the tube. Fischl in a study of 19 cases arrived at the same conclus the poor results were observed chiefly during the first trial, I knowledge that no extensive test was made by me, so that a defin ment regarding the method is at this time impossible. Cohnhe publication may induce me methodically to test the oil treatm But at present, on the basis of my previous experience, I must that in this treatment we possess no panacea for the symptoms in

Xeroform, one of the newer remedies (0.5 gram four times d been much praised. Jaworski advises, under the name of aq effervescens, a solution of sodium bicarbonate (8 or 5), sodium

(2½ or 2), and sodium biborate (2 or 1) in a liter of water, one-third to one-half of a tumblerful several times daily.

For the vomiting nothing is better than a carefully regulated diet. Large quantities of hot water may be taken several times daily, also pellets of ice with chloroform. Tincture of iodin (15 drops in 150 of water) has been advised by some authors. Special care is necessary if *hematemesis* appear; when very profuse this is self-evident, but it is also enjoined when smaller hemorrhages take place. Under all circumstances, the first requirements are absolute bodily and mental rest and the avoidance of all internal and external irritation of the stomach. Even in smaller hemorrhages, if circumstances permit, the patient should be restricted to this régime for several days, and the fullest precautions should be taken because these small hemorrhages are very frequently only the precursors of more profuse ones. Small pellets of ice, ice-cold tea, or ice-cold solutions of peptone in spoonful doses may be given. I do not give milk in such cases unless I know that the patient takes it well, but for the first day order either a solution of grape sugar to which some meat peptone bouillon is added and given ice-cold, or I administer cold, gelatinous soups of barley or oatmeal gruel; where the circumstances permit, nutritive enemata are cautiously given. The fluid extract of ergot, 2.5 to 5 of water and glycerin in equal parts, is subcutaneously injected in the gastric region, one to two syringefuls several times daily, but it must be remarked that ergotin in some persons gives rise to very unpleasant symptoms of constriction and vertigo. I have been unable to convince myself of the reliability of the fluid extracts of *hydrastis canadensis* and *hamamelis virginica*, which have lately been much used. When there is great irritability of the stomach, the injections of ergot should be combined with injections of morphin. This will usually arrest hemorrhage, provided large vessels are not implicated. Formerly lead acetate, iron chlorid, and oil of turpentine were employed internally because of their presumable styptic influence, but these are no longer used because we possess a more rational and active remedy in ergot. In some instances in which hemorrhages recurred for several days and the above measures were futile, the bleeding ceased upon *lavage of the stomach with ice water*. After preceding cocainization and the injection of a small quantity of morphin, the stomach-tube was carefully introduced, and the stomach wall repeatedly sprinkled with ice water, large amounts of hemorrhagic gastric contents being first washed out, whereupon the hemorrhage immediately ceased. Before proceeding to operate in such cases, this maneuver should always be first tried. It is occasionally astonishing to see what large quantities of hemorrhagic fluid are evacuated from the stomach. As some blood usually passes into the intestine and is there decomposed, and perhaps may set up irritative phenomena, it is advisable, if there is no spontaneous passage, to employ mild evacuants, preferably rhubarb with sulphur, or enemata; if symptoms of collapse appear, injections of camphorated oil (1: 6), enemata of wine, or wine with egg or peptone, and hot applications to the extremities. With threatening hemorrhage, a very small pulse, anemic murmurs over the heart, or cerebral anemia, normal saline infusions are indicated. Subcutaneous infusions are best given by employing a rather large Pravaz cannula. If the salt solution (7.5: 1,000) warmed to the temperature of the body is injected simultaneously through two needles, and the fluid introduced is disseminated by gentle friction, one liter of water may be introduced in a very short time. We prefer the subclavicular region as the point in which the needle should be introduced. In favorable cases the blood is rapidly regenerated. In a patient, aged 25, who had received an infusion, the blood upon the next day showed 2,100,000 erythrocytes, two weeks later 3,560,000, with a slight increase of the leukocytes.

Adrenalin or suprarenal extract has also been advised for gastric hemorrhage. Fenwick employs 1.3 grams of the dry glandular substance boiled in 230 of water. Internally or subcutaneously it is more rational to employ adrenalin hydrochlorid in solutions of 1: 1,000, 20 to 30 drops internally three to four times daily, subcutaneously 0.5–1 c.c. several times daily. There are quite a number of reports (Roussel, Renon and Louste, Mills, Kirch, Mamlock and others) in praise of its action, and authors are at least unanimous on the point that no unpleasant sequelæ (glycosuria, marked increase of blood pressure) appear even after its use for weeks.

Up to the present, I have employed the remedy internally in two cases, and it failed entirely. I must also say the same of *gelatin,* of which I gave a 10 per cent. solution up to 100 c.c. by mouth twice daily (it may also be used by enemata). In one case the hemorrhage continued, in two other cases, where the bleeding was but slight, this ceased with the simultaneous application of an ice bag to the gastric region and the swallowing of ice pellets. But who can say of such hemorrhages whether they cease spontaneously or under the influence of a drug!

Perforative peritonitis necessitates opium in large doses in the form of a suppository or as an enema, and the use of cold in the form of ice-cold compresses about the abdomen. If we suspect that the stomach is full, we must first try to empty it by the tube after either giving the patient a large dose of morphin or by applying cocain locally. But here it is necessary, under all circumstances, to keep the patient from retching, which may perhaps even enlarge the perforative opening. By this treatment it has sometimes been possible to limit the peritonitis locally, and to bring about adhesions. Operative measures have lately been advised for such cases, and several successful laparotomies have been reported (see below).

Surgical Measures.—For the florid ulcer, as well as for those with recent cicatrices, *surgical aid* has been invoked. Among the first to favor this were Rossoni in Rome, Nissen in St. Petersburg, and v. Mikulicz in Breslau. At the Surgical Congress (1897) the last author enumerated the indications as follows: "1. When the life of the patient is directly or indirectly threatened by hemorrhage, perforation, inanition. 2. When continuous treatment produces no, or but a temporary, relief, and the sufferings of the patient make his life miserable."

These indications may be somewhat more accurately defined, and operative measures resorted to under the following circumstances:

1. *In stenoses of the pylorus and hour-glass stomach due to cicatricial contraction,* or when continuous internal treatment is powerless to remove the symptoms. This presupposes the consideration that all internal remedies have been thoroughly and exhaustively employed. At the International Congress at Moscow in 1899 I gave the preference to this method of treatment in the but slightly developed field of gastric surgery rather than to operations for cancer, and ascribed to it the most satisfactory and the most lasting results.

2. In *perigastric adhesions* of the stomach to the surrounding organs. Here those adhesions of the greater curvature and at the pylorus are especially to be considered which by volvulus and torsion of the organs and the neighboring structures produce unbearable pain, all anodynes being ineffectual, while an operation at once removes the difficulties which may have existed for years. It is very surprising to see how delicate these adhesions sometimes are. In a case of ours recently operated upon the thin layers of the greater curvature led to the transverse colon, and could be almost completely detached without hemorrhage. Notwithstanding this, they must have been the cause of pains for months, since the patient had no symptoms after the operation.

3. *In gastric hemorrhage.* While the previously mentioned indications are clear and beyond question, provided the diagnosis is positive, in hematemesis it is extremely difficult to say whether and when operative measures are indicated. For even very threatening and very massive hemorrhages are apparently promptly arrested by proper internal treatment. Thrombosis appears with decreasing cardiac power, and it is astonishing to see how rapidly these exsanguine patients recuperate. v. Leube found uncontrollable hemorrhage to be the cause of death in 1 per cent. of his cases. Personally I have never seen a patient succumb immediately to hemorrhage. When Brinton claims 5 per cent. of fatal cases in ulcer due to hemorrhage, only the subsequent consequences, but not immediate death from hemorrhage, can be meant. This makes the decision as to operation difficult, as the results of surgery are by no means brilliant.

Often it is impossible to find the bleeding vessel at the operation, and there are cases in which this cannot be discovered even at the autopsy. Statistics are always very unreliable, for, according to the nature of the case, the successful operations, but not all of the unsuccessful ones, are published. Kaupe, the most recent author, collected 16 cases up to in which operation was performed because of acute life-threatening h rhage, and 10 of these were discharged as cured; this gives a mor of only 37 per cent., which figure decidedly differs from the actual The prospects would, perhaps, be more favorable if it could be pr and to this surgeons have recently called attention, that gastroenteros arrests the hemorrhage, that is, prevents its recurrence, without rea the actual source of the bleeding. Of course those cases only ca considered in which operation was performed during the hemorr for, under other circumstances, we can never be sure that the hemor did not cease spontaneously. Petersen and Machol actually state: have reached the positive conclusion that gastroenterostomy with Mur button might save many a patient with severe gastric hemorrhage would be lost under conservative treatment as well as by any other m of operation." The decision whether Murphy's button is actually a *ditio sine quâ non* for the successful result of the operation I must to the surgeons. But, as this technic has been mentioned in the quoted, I cannot refrain from raising the question (although from ciple I do not interfere in the technic of surgical treatment, and d usually permit myself an opinion) whether, for example, in the ca question, gastroenterostomy antecolica or gastroenterostomy retrocol indicated.

During the last few years, i. e., while Murphy's button was still use of by our German surgeons, I witnessed many operations wit without it, and I am convinced that its advantage of a more simpl therefore more rapid, technic does not compensate for its well know advantages. We are never sure that the button will not fall back in stomach, and in pyloric stenosis its fate is indefinite; we do not whether the narrow opening will functionate sufficiently, whether th ton has actually passed or has remained in the intestine (its passag have been overlooked by the nurses), and, provided everything go until then, it may even happen that the button is passed some week accompanied by very alarming symptoms which cause the greatest to the patients and also to the physician not sufficiently famili these conditions (for example, after the patient has left the hospital of this is obviated in a simple suture anastomosis which, in skilful consumes but little more time than the use of the Murphy button

4. A further indication for operative relief, and particularly troenterostomy, is given by *persistent hyperacidity,* especially if c with dilatation of the stomach and weakness of the expelling musc of course, only in those cases in which internal remedies have ineffectual after a thorough trial.

This occurs in connection with the *sequels of gastroanastomos* may influence the function of the stomach in various ways. W suppose, *a priori*, that when anastomosis is produced in the deepest area of the stomach a perfectly regular propulsion of the food would result. Some authors, for example, Rosenheim, go so far as to *assume the formation of a new sphincter.* The latter, however, as Petersen correctly remarks, must first be anatomically proven, and this has never yet been done. The possibility of closure of the anastomosis, which has been observed by Rosenheim, Carle and Fantino, or, more correctly, the fact that either water or air is retained in the stomach, merely proves that occasionally a valve-like closure of the opening occurs, not, however, that a sphincter is formed. On the other hand, the consensus of opinion is, and I have often seen convincing proof of this, that the propulsion of the gastric contents after operation is more rapid than before. Certainly if this were not so the result of the operation would be most unfortunate! Whether this is due to an increase of motility or to a freer outflow can hardly be decided. The absence of engorgement of the duodenum has been regarded as indicating an increased motility, for, according to Hirsch and v. Mering, normally, when the duodenum is full, it exerts an inhibitive reflex upon gastric movement. In this theory the fact has been overlooked that in by far the majority of cases in which gastroenterostomy is performed, stenosis of the pylorus occurs as well as a decrease or even a suspension of the propulsion of the ingesta from the stomach into the duodenum. Whether it is advisable in an open pylorus, as Kelling proposes, to close the passage from the stomach into the duodenum by the artificial production of stenosis, and thus prevent the conditions which have just been pointed out, can only be learned from surgical experience, reports of which are still lacking.

There is no doubt that, in many cases, *improvement in the chemism* of the stomach has been brought about. The acidity value, more accurately the value for the excretion of hydrochloric acid, decreases, perhaps less in consequence of decreased secretion than because the bile which regurgitates into the stomach, possibly also the pancreatic juice, neutralizes the acid gastric contents. But that such a decrease of acidity does not take place in all cases may be adduced from my own experiences, which, unfortunately, are not yet published, as well as from the reports of numerous other authors, Dunin, Oderfeld, Kausch, Rosenheim, Rencki, Petersen and others. Nevertheless, the decrease of acidity is so frequent that the above indication for gastroenterostomy in hyperacidity must be mentioned. The regurgitation of bile and pancreatic juice into the stomach—which, under the present method of performing enterostomy between the loops leading to and away, is now usually prevented—gives rise to but slight, if any, difficulty, and this is only perceptible by a bilious taste. Experiments which Joslin undertook in the year 1897 at my suggestion revealed decreased fat digestion, but this, as Nikolaysen later found, is only immediately after the operation, and then is replaced by normal conditions. The experiments at that time, however, were made upon patients who had n been operated upon; what the results would be after this operation still to be determined.

5. In case of *perforation,* operation whenever possible should be pe formed in the first twenty-four hours; the later, the worse the chanc In 1895 Pariser could report only 43 cases.

In the year 1896 Weir and Foote compiled a table of the cases perforating gastric ulcer which had been operated upon, and this show 78 cases with 23 recoveries, = 29 per cent. In 43 cases the perforati opening was situated upon the anterior wall of the stomach, 11 times up the posterior wall, and 6 times on the lesser curvature. Accordingly, in t majority of cases this opening may be found and sutured. In 92 per ce of the cases the history pointed to the diagnosis of gastric ulcer. T importance of early operation is very evident, for in the cases that h existed less than twelve hours the mortality was 39 per cent., in those twelve to twenty-four hours 76 per cent., and in those over twenty-fo hours 87 per cent.

The number of such cases rapidly increased. In 1900, Mayo Robs published statistics compiled from reports of English and American h pitals, comprising 429 cases with a mortality of 55 per cent.; Brunn however, declares that some cases were counted twice. In the year 19 Brunner compiled the statistics of 466 cases upon whom operation f gastric perforation had been performed, which gave a mortality of 50 64 per cent.; here also the advantage of early operation was clearly demo strated, for, among the cases in which operation was performed in the fi twelve hours, 75 per cent. recovered! It is well known that the chanc of recovery for the patient are much better if the stomach at the ti of perforation is empty.

Unfortunately, under some circumstances, we cannot determine wheth in the case in question we are dealing with a perforative peritonitis due ulcer of the stomach or not. I have seen a case of this kind terminate recovery, yet during its course and even later no positive diagnosis w possible.

Brunner collected reports of 17 cases recovering without operation, against 466 who were operated upon in the same period of time. T number, 17, has been increased by a recent English report of a few ca (*British Med. Journal,* Feb. 20, 1904, H. Whiteford), which, howev is not quite trustworthy.

6. By some authors the *pure, uncomplicated ulcer* has also been des nated as suitable for surgical treatment. I do not agree with this. uncomplicated ulcer heals readily under internal treatment; if heal does not thus take place the ulcer is not uncomplicated, but is combi with changes which resist the regenerative process. Under such circ stances, the indication for operation has been assumed from the fact t

n ulcer may occasionally undergo malignant degeneration. This is anal-gous to the proposition to operate upon every appendix in which there re signs of inflammation, because subsequently perforation might result. ut in the one instance, as in the other, this is but a remote possibility, nd in the case of ulcer the danger is very slight, for the number of cases ured decidedly preponderates over those of uncured ulcer of the stomach. s shown above, this proportion is about 2 : 1, but in reality only a small ractional part of unhealed cases undergo malignant degeneration.

Finally, as a general indication for operative interference, immaterial hich of the conditions enumerated comes into question, it must be borne n mind that operation only removes the existing pathologic focus, not the redisposition to future relapses. Therefore, the appearance of a new lcer, another hemorrhage, fresh adhesions, is by no means impossible fter a successful operation, and Brasch has described a case in which atal hemorrhage occurred immediately after gastroenterostomy had been erformed. *Consequently those surgeons are right who proceed to operate nly when all the remedies of internal treatment have been exhausted, hat is, when the condition of the patient permits no further delay. That ases of acute perforation are exceptions to this is self-evident.* In his pinion, and also in the warning against exaggeration of the indications for operation, as expressed by Sahli before the Twentieth Congress of Internal Medicine in 1902, I concur almost absolutely.

It should never be forgotten that the process of healing after these operations is not always smooth and unbroken. I do not refer to the danger of the so-called vicious circle, i. e., the regurgitation of the gastric ontents into the false intestinal loop in gastroenterostomy, because this perhaps is avoided by the simultaneous enterostomy, but I must call attention to the occasional development after operation of peptic intestinal ulcers which are a new source of difficulty and danger to the patient. Neumann has recently reported a very instructive case of this kind, and in my opinion given very valuable advice, which is, that in gastric dilatation with pylorospasm and hyperacidity we should first perform jejunostomy and then allow the stomach to rest. Later, if necessary, and under favorable circumstances, gastroenterostomy may be performed.

Mineral Spring Treatment.—Since remote times the hot Glauber Salt Springs, particularly those of Carlsbad, have enjoyed a well deserved reputation, and there is no doubt that the treatment of ulcer at this resort, when carefully conducted and too large quantities of water at one time are not consumed, is frequently crowned by the best results. When the disturbances on the part of the digestive apparatus are relieved, the patient should endeavor to recuperate and gain strength by a sojourn at Franzensbad, Elster, Rippoldsau, Pyrmont, etc., in the mountains or at he sea, with the proviso that he can secure suitable food, which is best hen the family does its own cooking. Since, however, many patients prefer a mineral spring cure to going to bed at home, and many avail themselves of the short period of four to six weeks, Carl these is always the best place, because the opportunity there for sins is less than elsewhere. After Carlsbad, Neuenahr, Ems, Fran Homburg and Vichy are to be recommended.

LITERATURE

Agéron, "Diagnostisch-therapeutische Bemerkungen zum Magengeschwür." *ener med. Wochenschr.*, 1902, Nr. 30.

Alberti, *Deutsche Wochenschr.*, 10. Januar, 1901, Vereinsbericht.

O. Ashe, "Excision of a Perforated Gastric Ulcer; Recovery." *Brit. Me* December 15th, 1903.

Backmann, "Verbreitung des runden Magengeschwürs in Finnland." *Z klin. Med.*, Bd. XLIX.

F. Blumensath, "Statistisch-klin. Mittheilungen über das runde Magenge *Inaug.-Dissert.*, Kiel, 1902.

Boas, "Ueber occulte Magenblutungen." *Deutsche med. Wochensch* Nr. 20.

Borrmann, "Das Wachsthum und die Verbreitungswege des Magencar Jena, 1901, G. Fischer.

L. Bourget, "Therapie des Ulcus ventriculi." *Therap. Monatsh.*, Juli, 19

Brinton, "Die Krankheiten des Magens." Uebersetzt von H. Bauer, W 1862.

Fr. Brunner, "Das acut in die freie Bauchhöhle perforirende Magen- und algeschwür." *Deutsche Zeitschr. f. Chir.*, Bd. LXIX, p. 101.

Byrom Bramwell, "On Gastric Ulcer." *Lancet*, March 9th, 1901.

Cabot, "Indications for Operation in Gastric Ulcer." *Boston Med. Jou* Nr. 9.

Carle und *Fantino*, "Beiträge zur Pathologie und Therapie des Magens. *f. klin. Chir.*, 1898, Bd. LVI.

P. Cohnheim, "Heilwirkung grosser Dosen von Olivenöl." *Zeitschr. f. kl* Bd. LII, Heft 1 und 2.

Cesaris Demel, "Syphilitisches Magengeschwür." *Wiener med. Press* Nr. 8.

Dolmatow, "Zur Frage über den Werth der Verdauungsleukocytose be carcinom." (Polish.) Jahrber., 1900.

M. Einhorn, "Beitrag zur Kenntnis und Behandlung der Erosionen des *Berliner klin. Wochenschr.*, 1895, Nr. 20, und *Arch. f. Verdauung* Bd. V, p. 317.

H. Elsner, "Zur Frage der hämorrhagischen Erosionen des Magens." *med. Wochenschr.*, 1903, Nr. 41.

Eppinger, "Ueber Tuberculose des Magens und Oesophagus." *Pra Wochenschr.*, 1881, Nr. 51 und 52.

C. A. Ewald, "Diagnose und Therapie des Magengeschwürs." *XX. innere Med.*, Wiesbaden, 1902. *Ibid.*, Nr. 23, p. 347. "Ueber die des Ulcus ventriculi mittels Nachweises occulter Blutanwesenhe Fäces." *Deutsche med. Wochenschr.*, 1903, Nr. 47. *Berliner klin. W*

1888, p. 396. "Zur Diagnose des Sanduhrmagens." *Deutsches Arch. f. klin. Med.*, Bd. LXXIII, p. 152.

Eysenhardt, quoted by Struppler.

Fenwick, "Suprarenal Extract in Gastro-intestinal Haemorrhage." *Brit. Med. Journ.*, November 30th, 1901.

Frz. Fink, "Erfolge der Karlsbader Cur u. s. w." Wien und Leipzig, 1903.

L. Fischl, "Zur Therapie der Hyperacidität des Magens." *Prager med. Wochenschr.*, 1903, Nr. 11.

Fleiner, "Therapie des Magengeschwürs." *XX. Congr. f. innere Med.*, Wiesbaden, 1902.

Friedenthal, "Ernährungsschwierigkeiten bei Ulcus ventriculi." *Inaug.-Dissert.*, Berlin, 1899.

Futterer, "Treatment of Chronic Round Ulcer of the Stomach." *Journ. Amer Med. Association*, January, 1902.

Gaillard, "Syphilis gastrique et ulcère de l'estomac." *Arch. génér. de méd.*, 1886.

D. Gerhardt, "Ueber geschwürige Processe im Magen." *Virchow's Arch.*, Bd CXXVII, p. 85.

Gluczinsky, "Ueber die Behandlung des peptischen Magengeschwürs." *Wiener klin. Wochenschr.*, 1900, Nr. 49.

Greenough and *Joslin*, "Gastric Ulcer at the Massachusetts General Hospital." *Amer. Journ. of Med. Sciences*, August, 1899.

Hampeln, "Gastro-intestinale Blutungen." *St. Petersburger med. Wochenschr.*, 1891, Nr. 8.

Harttung, "Ueber Faltenbildung und hämorrhagische Erosionen." *Deutsche Wochenschr.*, 1890, p. 847.

Hemmeter, "Zur Histologie der Magenschleimhaut." *Arch. f. Verdauungskrankh.*, Bd. IV, p. 24.

A. Hirsch, "Beiträge zur motorischen Function des Magens." *Centralbl. f. klin Med.*, 1892, Nr. 47.

P. Hirschfeld, Discussion. *XX. Congr. f. innere Med.*, Wiesbaden, 1902.

W. C. Hood, "Hæmatemesis with Special Reference to that Form met with in Early Adult Female Life." London, 1892.

Kausch, "Ueber functionelle Ergebnisse nach Operationen am Magen." *Grenzgebiete*, Bd. IV, p. 347.

G. Kelling, "Sympathischer Reizzustand bei Magengeschwür." *Wiener med. Wochenschr.*, 1902, Nr. 48.

Kocher, "Indicationen zur Operation bei Ulcus ventriculi." *Correspondenzbl. f. Schweizer Aerzte*, 15 October, 1898.

G. Köhler, "Beitrag zur Kenntnis der Symptomatologie bei Ulcus ventriculi simplex." *Inaug.-Dissert.*, Berlin, 1895.

Körte, "Chirurgische Behandlung des Magengeschwürs." *Arch. f. klin. Chir.*, Bd. LXIII, p. 1.

Krafft, "Beitrag zur Pathogenese des Ulcus ventriculi." *Hospitalstidende*, 1900.

Krönlein, "Ueber Ulcus und Stenosis des Magens nach Trauma." *Arch. f. Chir.*

L. Kuttner, "Magenblutungen und deren Beziehungen zur Menstruation." *Berliner klin. Wochenschr.*, 1895, Nr. 7.

Lange, *Deutsche Klinik*, 1860, p. 90.

Langerhans, "Ungewöhnliche Art der hämorrhagischen Erosionen des Magens." *Virchow's Arch.*, Bd. CXXIV, p. 373.

Letulle, *Compt. rend.*, 1888, Vol. CVI.

v. Leube, "Ueber die Erfolge der internen Behandlung des Magengeschwürs und die Indicationen zum chirurgischen Eingreifen in dieselbe Grenzgebiete," Bd. II. *Referat auf dem XXVI. Chirurgen-Congress*, 1897.

Leuk, "Untersuchungen zur pathologischen Anatomie des menschlichen Magens," etc. *Zeitschr. f. klin. Med.*, 1899, Bd. XXXVII.

Litten, "Ulcus ventriculi tuberculosum." *Virchow's Archiv*, Bd. LXVII, p. 615.

L. Lorell-Keays, "A case of Double Perforating Gastric Ulcer." *Brit. Med. Journ.*, December 15th, 1903.

Luxenburg und *Jawadzki*, "Ein Fall von Ulcus ventriculi rotundum auf Grund syphilitischer Gefässerkrankung." *Wiener Med. Presse*, 1894, Nr. 50 und 51.

J. N. Marshall, "Two Cases of Gastric Ulcer in which Symptoms arose suggesting Perforation of the Stomach." *Glasgow Med. Journ.*, February, 1890.

A. Mathieu, "Traité des maladies de l'estomac et de l'intestin." Paris, 1901.

Mathieu et *Roux*, "Sur un cas d'ulcérations urémiques de l'estomac et de l'intestin grêle." *Arch. gén. de méd.*, Janvier, 1902.

v. Mering, "Zur Function des Magens." *Congr. f. innere Med.*, Berlin, 1897.

H. Merkel, "Ein Fall von chron. Magengeschwür mit tödtlicher Blutung aus der arrodirten linken Nierenvene." *Virchow's Archiv*, 1903, Bd. CXXIII.

E. Mey, "Profuse Magenblutungen und Hydrops anasarca als initiale Symptome des Magencarcinoms." *Inaug.-Dissert.*, Dorpat, 1891.

Michailow, "Ein Fall von erfolgreichem chirurgischen Eingriff bei Blutungen aus einem Magengeschwür." *Grenzgebiete*, 1901, p. 535.

v. Mikulicz, "Die chirurgische Behandlung des chron. Magengeschwürs." *Berliner klin. Wochenschr.*, 1897, Nr. 23.

Minkowski, *Discussion. XX. Congr. f. innere Med. zu Wiesbaden*, 1902.

S. Mintz, "Hämorrhagische Magenerosionen." *Zeitschr. f. klin. Med.*, Bd. XLVI, Heft 1 to 4.

Marfan, "Troubles et lésions gastriques dans la phthisie pulmonaire." Paris, 1887.

William Murrell, "The Value of Age and Sex as Etiological Factors," etc. *Med. Press and Circular*, October 23d, 1901.

J. H. Musser, "Tubercular Ulcer of the Stomach." *Philadelphia Hosp. Reports*, 1890, Vol. I.

O. Müller und *Hecker*, quoted by Struppler.

Nauwerk, "Mykotisch-peptisches Magengeschwür." *Münchener med. Wochenschr.*, 1895, Nr. 38 und 39.

v. Openchowski, "Zur pathol. Anatomie der geschwürgen Processe im Magen-Darmtractus." *Virchow's Archiv*, Bd. CXVII, p. 347.

W. Ord, "On the Diagnosis and Treatment of Gastric Ulcer." *Amer. Journ. of Med. Sci.*, June, 1889.

Osterspey, "Die Bluntuntersuchungen bei Magenkranken." *Berliner klin. Wochenschr.*, 1892, Nr. 12 und 13.

Pariser, "Discussionsbemerkungen im XX. Congr. f. innere Med. zu Wiesbaden. Ueber hämorrhagische Erosionen der Magenschleimhaut." *Berliner klin. Wochenschr.*, 1900, Nr. 43.

Paulicki, Virchow's Archiv, Bd. XLIV.

Petersen und *Machol*, "Beiträge zur Pathologie und Therapie gutartiger Magenkrankheiten." *v. Bruns' Beiträge zur klin. Chir.*, Bd. XXXIII, p. 297.

Petruschky, "Zur Diagnose und Therapie des primären Ulcus ventriculi tuberculosum." *Deutsche med. Wochenschr.*, 1899, Nr. 24, und 1901, p. 394.

N. Platter, "Ueber Erosionen der Magenschleimhaut." *Inaug.-Dissert.*, Zürich, 1901.

Reichard, "Freie Vereinigung der Chirurgen Berlins." *Deutsche med. Wochenschr.*, 1890, p. 327.

R. Reinhard, "Ulcus ventriculi simplex mit Tumoren." *Inaug.-Dissert.*, Berlin, 1888.

R. Rencki, "Diagnostische Bedeutung der mikroskop. Blutuntersuchung bei Carcinom und Ulcus ventriculi." *Arch. f. Verdauungskrankh.*, 1901. "Ueber die functionellen Ergebnisse nach Operationen am Magen bei Ulcus und gutartiger Pylorusstenose." *Grenzgebiete*, 1901, Bd. VIII.

Rheinwald, "Die Behandlung des einfachen Magengeschwürs mit Karlsbader Curen." *Inaug.-Dissert.*, Tübingen, 1898.

F. Riegel, "Beiträge zur Diagnostik der Magenkrankheiten." *Zeitschr. f. klin. Med.*, Bd. XII, Heft 5.

Mayo Robson, "A Clinical Lecture on the Complications of Gastric Ulcer and Its Treatment." *Brit. Med. Journ.*, February 2d, 1901.

Rosenheim, "Ueber seltene Complicationen des runden Magengeschwürs." *Berliner klin. Wochenschr.*, 1889, p. 1031. "Ueber die chirurgische Behandlung der Magenkrankheiten." *Deutsche med. Wochenschr.*, 1895, Nr. 1.

Schloffer, "Operationen bei gutartiger Magenerkrankung." v. Bruns, *Beiträge zur klin. Chir.*, Bd. XXXII, Heft 2.

A. Schmidt, "Pathogenese des Magengeschwürs." *Verhandl. XX. Congr. f. innere Med.*, Wiesbaden, 1902.

Schütz, "Zur Differentialdiagnose des Ulcus ventriculi." *XVII. Congr. f. innere Med.*, Wiesbaden, 1899.

J. Schulz, "Ueber Dauererfolge der internen Therapie des Ulcus ventriculi." *Grenzgebiete*, 1903, Bd. XI.

Schwarz, "Beiträge zur Pathologie und chirurgischen Therapie des penetrirenden Magengeschwürs." *Grenzgebiete*, 1900.

J. Sigel, "Zur Diagnose des Magencarcinoms." *Berliner klin. Wochenschr.*, 1904, Nr. 12 und 13.

M. Silbermark, "Rundes Magengeschwür nach Trauma." *Wiener med. Wochenschr.*, 1902, Nr. 21 und 22.

Simmonds, "Ueber Tuberculose des Magens." *Münchener med. Wochenschr.*, 1900, Nr. 10.

v. Sohlern, "Einfluss der Ernährung auf die Entstehung des Magengeschwürs." *Berliner klin. Wochenschr.*, 1889, Nr. 13 und 14.

Spicker, "Spontan geheilte Perforationsperitonitis bei Ulcus ventriculi." *Deutsche med. Wochenschr.*, 1903, Nr. 1.

Sepp, "Zur Behandlung des chron. Magengeschwürs." *Festschr. d. Nürnberger ärztl. Vereins*, 1902.

Stern, "Ueber traumatische Erkrankungen der Magenschleimhaut." *Deutsche med. Wochenschr.*, 1899, Nr. 23.

H. Strauss und *Bleichröder,* "Untersuchungen über den Magensaftfluss." *Gre gebiete,* 1903, Bd. XII, Heft 1.

Struppler, "Ueber das tuberculöse Magengeschwür." *Zeitschr. f. Tuberculose u Heilstättenwesen,* Bd. I, Heft 4.

Talamon–Balzer, "Phthisie locale; ulcérations tuberculeuses de l'estomac et l'intestin." *Bullet. Soc. Anatom.,* 1878, p. 374.

S. Talma, "Untersuchungen über Ulcus ventriculi simplex." *Zeitschr. f. k Med.,* Bd. XVII, Heft 1 und 2.

M. Tiegel, "Beitrag zur Casuistik tödtlicher Magenblutungen." *Münchener n Wochenschr.,* 1902, Nr. 47.

Dalla Vedova, "Experimenteller Beitrag zur Kenntnis des Ulcus ventriculi." *A f. Verdauungskrankh.,* Bd. VIII, Heft 3.

Walko, "Ueber die Behandlung des Ulcus ventriculi mit Olivenöl." *Centr f. innere Med.,* 1902, Nr. 45.

M. Weiss, "Magenblutung bei Typhus abdominalis." *Wiener Med. Presse,* 1

Frz. Warnecke, "Indicationen zur operativen Behandlung des Ulcus ventric Preisschr., Göttingen, 1903.

van Yzeren, "Die Pathogenesis des chron. Magengeschwürs." *Zeitschr. f. Med.,* Bd. XLIII, p. 181. (Also see the literature in Ewald's article "M krankheiten," *Eulenburg's Real-Encyclopädie,* 1897, Bd. XIV.)

GASTRIC AND INTESTINAL CARCINOMATA

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When a case of gastric or intestinal carcinoma presents itself before us to-day, we judge it by very different standards from those employed about twenty years ago. At that period, i. e., in the days of Billroth, Péan and Rydygier, our chief endeavor was to make the *diagnosis,* and at that a diagnosis of general visceral cancer. Whether the cancer was located in the stomach and in what part of the same, whether in the small or large intestine, whether in the omentum or the liver, in the gall-bladder or the pancreas, was of scientific but not of therapeutic interest. In every case the patient was doomed; to him accurate proof that this or that portion of his viscera showed malignant degeneration was of no avail.

In the last two decades our views concerning these affections have undergone a complete change the influence of which no physician, however pessimistic he may be regarding carcinoma in general, can withstand. This change, as is well known, has been brought about by *surgery,* and no genius has yet appeared to teach us how to cure by internal remedies. The knife alone can permanently remove the difficulty, or, although very rarely, bring amelioration which lasts for years.

This advance, which is largely due to the high development of antisepsis and asepsis, but also to an enormous impetus to the technic of abdominal surgery, imposes upon internal medicine new obligations.

The diagnosis must be made early. It must accurately define the seat of the affection, it must—when possible—embrace the consideration whether the malignant focus is circumscribed or already generalized. It must decide whether or not any complications are present which may influence the course of the operation, and, finally, it must determine whether at the given moment the patient's strength will warrant surgical interference.

If it were possible in a case of gastric or intestinal carcinoma to decide all these points with certainty, the results of operative treatment would be decidedly more favorable than they are at present, and this would doubtless influence strongly our decision as to conservative or active treatment. Although, in fact, this goal is still remote, our chief endeavor must be to arrive as near as possible to the previously mentioned postulates. For this purpose not only a comprehensive diagnosis, but also the earliest recognition of the affection from all points of view is necessary.

After these preliminary remarks I shall describe a typical case of tric carcinoma, the delineation of other less typical forms will follow and I shall then describe a case of carcinoma of the large intestine of the rectum; for cancer of the small intestine, on account of its ra a brief description will suffice; in conclusion I shall summarize the t ment of gastrointestinal carcinomata.

ETIOLOGY

In examining the ***history*** of this patient it will be noted that h about 50 years of age, and up to the time of his present affection he usually been well; above all, he has had no gastric affection. Until wi the last few months he could eat anything and digest it. His pre illness dates from this period.

Let us here pause for a moment, since these data are of great import as indicating the character of the affection. When a patient, who reached the age at which cancer is likely to occur, tells us that he never been ill, while his appearance denotes the existence of a se malady, we must primarily think of a malignant disease. Not that report is in itself of vital importance, but it indicates the directio which we must search for the underlying affection.

We have heard from this patient how his ailment gradually deve to its present extent. We are especially interested in learning wha first symptoms were, and whether these gave any indication of the g of the condition.

His report, in brief, is the following: On taking food, he has nov then a sensation of pressure which is not actual pain in the pit c stomach, occasionally there is eructation of gas but not of food, and this a gradually increasing loss of appetite. This is about all. W the justification of Brinton's expression in regard to the onset of g carcinoma: "Obscure in its symptoms." Nevertheless, any one wh had much experience in the realm of visceral carcinomata will not the conviction that this latent and indefinite onset forms the rule to there are, however, numerous exceptions of practical importance. these belongs the *sudden onset with severe symptoms*: In the mi apparently normal health the patient begins to vomit, and to the s of those about him, even of the physician, this continues and s becomes more copious so that in a few days, or at most in a few the patient emaciates markedly, loses his appetite, and becomes feel debilitated; briefly, he shows such extreme emaciation that, provi severe functional or central affection is present—in this connection particularly to the gastric crises and the periodical vomiting desc v. Leyden—without more ado the suspicion of malignant disease is a

Another mode of onset of practical significance is when gast cinoma is most surprisingly ushered in by decided and threatening *hematemesis* or *melena*. This naturally leads us to think of a simple ulcer of the stomach or of the duodenum, and if etiologic factors are favorable, perhaps also of cirrhosis of the liver or some other form of disease leading to stasis of the portal vein; but the further course shows that a scarcely avoidable error has been made. In connection with the hemorrhage isolated symptoms occur, or all the symptoms of a malignant course may be required to make the diagnosis of cancer more positive from day to day. Practically we may easily assume that a gastric ulcer is undergoing malignant degeneration, but it has been proven with certainty (by May, Ewald and others) that such a course by no means always permits the conclusion of a preceding ulcer. I shall refer to this again in the description of carcinomatous ulcer.

But such occurrences as have been mentioned are usually exceptional. The typical onset, as we unfortunately know, is usually so slightly characteristic that the last thing the patient and his relatives think of is the development of a malignant, incurable disease. And thus it happens that at first, according to their degree of education, they become their own physicians and employ all sorts of home remedies and occasionally purgatives up to a period when every effort at internal or surgical treatment is hopeless. But with careful and comprehensive professional treatment from the first, all concerned will have an expectant attitude. The wise physician will reckon on the possibility of a beginning malignant affection, but because of the absence of definite symptoms, he will refrain from expressing his fears, and not cause unnecessary fright and distress.

At the onset, I purposely mention the difficulties in the recognition of the disease. It will be seen that the demand of many surgeons for an early diagnosis is scarcely justified, for we cannot make a diagnosis before we have the patient. In the chapter upon treatment we shall also consider the fact that, even with an early diagnosis, rapid surgical intervention is sometimes prevented by the stress of circumstances.

After this brief digression, we return to the further course of the disease as described by our patient. He states that in addition to the slight difficulties at the onset disturbances of more serious nature now and then occurred; there was pressure, also pain in the pit of the stomach which occasionally extended from the left side posteriorly into the back, radiating also to the shoulder-blade. Sometimes the patient mentions a symptom that, so far as known to me, is nowhere recorded—a peculiar grating in the gastric region as of two surfaces rubbing against each other. I have heard this symptom described in the same manner by several patients, so that in enumerating the subjective disturbances it appears to me to be of consequence. Now and then vomiting occurs, at first rarely and in slight amount, in the last few weeks more frequently and more copiously. The vomitus consisted of the ingested food which, in the first stages, showed no alteration in appearance, but lately it has now and then somewhat brownish. After vomiting the patient is temporarily rel At first he lost some flesh; in the last few weeks the weight has de rapidly, and hand in hand with this there has been a marked decre strength; the appetite is wholly lost.

This is the stage in which patients with carcinoma of the stc usually present themselves to the physician. Clear as the course c development appears, and strongly as the individual points favor a r nant disease, we must not forget that other severe but not actuall lignant processes may have the same or a very similar course of de ment. I refer, for example, to the cases, recently so accurately st of hypertrophic stenosis of the pylorus, the symptoms of which c resemble those just described. Cicatricial stenosis of the pyloru may remain latent for a long time, and only when, by a sudden and sional cause, the musculature is insufficient, may show symptoms very closely resemble in severity and extent closure by a malignant pr

Only an accurate clinical analysis of the case will decide wheth very suggestive reports of the patient find their explanation in the *ob examination.* While the patient was undressing it was noted th extraordinary extent of the disturbance in nutrition was most obviou the upper to the lower extremity. The musculature was soft and the subcutaneous connective tissue thin, atrophic and without fat, tl itself could readily be lifted in folds. The *tongue* was coated at and borders. From the present stand of gastric pathology too mu portance cannot be attached to the condition of this organ. factors here come into consideration (tobacco and alcohol, their r abuse, fissures, inflammation of the tonsils and pharynx, greater cleanliness of the oral cavity) so that the relation between gastr culties and the tongue cannot be accurately determined. In con Leser, I do not attach much importance to the small miliary a miliary angiomata which this patient also showed, for, in my exp which coincides with that of Gebele, this is not rare in non-carcin cases, particularly in the aged.

After the patient had removed his clothes he was systematic amined in the usual manner. To avoid taking up unnecessary shall state at once that the examination of the heart and lungs normal conditions.

In the examination of the *abdomen* I must promptly emph importance of *inspection of the abdomen,* for this method furnish ness of diagnostic aids which can only be recognized by experienc the configuration of the abdomen must be noted, and the devi both its halves carefully compared. To the right of the medi relief-like projection is then noted, a slight prominence, which inspiration descended, and upon expiration rose again to its old

I shall revert to this later. But another very remarkable phenomenon was noted! From time to time a second relief was seen in the left hypochondrium, rising suddenly and prominently, almost like a mound, from the level of the abdomen, and after a brief time sinking to its old position of rest.

While inspecting the abdomen carefully, and at the conclusion of this phenomenon, a peculiar gurgling murmur was heard.

What does this phenomenon mean? It will perhaps be said that it denotes peristaltic unrest of the stomach, as was first described in a classical manner by Kussmaul, or, as the French term it, "peristaltism." But if this sign which was found to recur at brief intervals and always to run the same course be more accurately examined, we may convince ourselves that it is not from true peristaltic action. On the contrary, it resembles a tonic contraction which does not develop further but remains localized.

The picture is so fascinating that when the phenomenon is at its height we cannot refrain from grasping the prominence with the hands. We feel a hard, contracted mass which resembles the uterus during a pain. No doubt this is the fundus of the stomach. We designate this process "*gastric rigidity,*" and this is analogous to the nomenclature Nothnagel proposed for similar processes in the intestine, to which we shall refer later.

This sign, to which I have recently again called attention,[1] was observed about fifty years ago by no less a one than Cruveilhier,[2] and I cannot refrain from quoting what he says of this symptom in his great "Traité d'anatomie pathol. générale," as follows:

"La dilatation hypertrophique de l'estomac s'observe dans les cas où l'ampliation de l'estomac se faisant graduellement, la contractilité et la force élastique de l'organe n'ayant pas été vaincues, il y a résistance à la distension de la part de la tunique musculeuse, qui se contracte énergiquement sur les aliments, les boissons et les gaz, qu'il contient. *Cette contraction qui se manifeste d'une manière intermittente est facile à reconnaitre pendant la vie par un durcissement douloureux analogue aux légères contractions utérines, qui précèdent les grands douleurs de l'accouchement. Pendant toute la durée de cette contraction la forme de l'estomac se dessine à travers les parois abdominales et chez les sujets amaigris on peut en suivre tous les contours à l'oeil et au doigt. C'est à mon avis un grand moyen de diagnostic.*"

In this description the process of gastric rigidity is so masterfully portrayed that little of importance can be added.

My own experience has taught me that several stages of gastric rigidity may be differentiated: 1. Slight contractions extending only to small areas of the fundus, which are felt as increased tonicity lasting but a few se (abortive gastric rigidity); 2. Distinct contractions conveying the tion of marked increase in tonicity, becoming visible as globular inences, being contractions of a greater or lesser area of the fundus terminating with a distinct pressure murmur which is felt by the p as a more or less painful contraction; 3. Decidedly marked, visibl palpable contractions in the entire extent of the gastric fundus, continue for a long time, terminate with a distinct gurgle, and pr active pain.

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[1] *Boas, Deutsche med. Wochenschr.*, 1902, Nr. 10.

[2] *Cruveilhier,* "Traité d'anatomie pathol. générale," 1852, T. II, p. 857.

This gastric rigidity is apparently the precursor of peristaltic u Its relation to the latter is that of tonus to tetanus. Occasionally, ticularly when the stomach is overloaded, both may be observed by side.

Gastric rigidity is in my opinion of very *decided diagnostic impor* It indicates at a relatively early stage that there is an obstruction pylorus. Of what nature this is, whether purely functional (s contraction of the pylorus) or organic, whether an ulcer, a neopla gastrolith, an adhesion of the pylorus to neighboring organs, volvulus can only be decided by continued objective analysis of the individual But the fact alone that the patient is suffering from an obstructi or near the pylorus is not only of great import in the diagnosis bu in the prognosis, and even in treatment.

For this purpose I wish to add a few remarks of practical signifi to the discussion of the symptom of gastric rigidity, which is not ciently considered in medical literature. "Gastric rigidity," for o reasons, may be best noted when the organ is full and it may then palpated; but, even then, it can occasionally only be observed with friction of the gastric region, preferably with a cool hand or a han has been dipped in ice-water. As gastric rigidity does not always even under these conditions the examination must be frequently re A slight degree of gastric rigidity can only be determined by practice, and I therefore advise that the students practise first wit in which the rigidity is well developed, so as to determine the in gastric tonus.

I have purposely dwelt upon this phenomenon at some length, f out a complicated apparatus it at once gives the physician reason pose that something is out of order in the motor apparatus of the s We have then a certain foundation upon which to proceed, and w what further steps must be taken to clear the situation.

We shall now recur to the previously mentioned visible *pro* We note that it shows inspiratory and expiratory motion, and proceed to palpate it. *As may be readily determined, we are deal a hard tumor of uneven surface and about the size of a small ora*

Apparently this completes the diagnosis. We have before us of the pylorus. But we must investigate still further to ascertain the nature of this tumor. Is it movable or adherent? Can it be fixed upon expiration or not?

The movability of a pyloric tumor (for only these can be decidedly moved manually) is so readily proven that we really need say nothing further in regard to it. But only a very few pyloric tumors are movable in all directions like the head of a child in the uterus; in the majority of cases there are some adhesions, and it is then necessary to determine the degree of movability.

If we are in doubt, it is well to inflate the stomach with carbonic acid or air, when it will be possible to determine the position of the stomach, especially that of the greater curvature. If while inflated by air the tumor is still movable, certain locomotor excursions downward and to the right will occur, occasionally also upward and to the right.

Under this method in the case in question we note that inflation by CO_2 (which we prefer on account of its simplicity and particularly because the patient has not yet become accustomed to the prolonged presence of the stomach-tube) affects the position of the tumor but little. Therefore, it is probably adherent to the surroundings.

At this point we must consider an important difference between the expiratory relation of tumors of the pylorus and tumors of the small curvature. The latter, as was first determined by Minkowski,[1] are characterized by *expiratory immovability*. By this sign tumors of the small curvature may readily be differentiated from tumors belonging to other organs, and on the other hand it is also an important factor in differentiating them from tumors of the pylorus. Yet, tumors of the small curvature may lose their expiratory immovability when they become adherent to neighboring organs, but in this case they either do not ascend with inspiration, or they slightly descend.

Judging from the previously mentioned findings the tumor in this case proves to be a tumor of the pylorus, and since it is hard to the touch and the other symptoms also point to a malignant course, we can hardly be wrong in deciding that we have before us a carcinoma of the pylorus.

Nevertheless, although we feel quite certain of our opinion, we shall complete as far as possible the physical examination of the organ, as well as of the other abdominal organs.

Previously, on inflating with CO_2 and simultaneously ascertaining the movability of the pyloric tumor, we observed the position of the greater curvature. It was noted under these circumstances that it extended above the horizontal umbilical line about a handbreadth. We were then unable to determine the position of the smaller curvature as this is only possible on inflating the stomach with air to the extreme limit. These methods, therefore, do not permit us to ascertain whether the stomach is enlarged or descended, or whether both conditions are present.

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[1] *Minkowski, Berliner klin. Wochenschrift*, 1888, Nr. 31.

If we desire accurate knowledge, we must resort to maximal inflation with air, filling with water, or, eventually, the illumination of the stomach. These tests may perhaps have a scientific value, but are of little practical importance.

It is more serviceable to investigate the organs adjacent to the stomach, especially the liver, the intestines, and the mesentery.

As the metastases of carcinoma are principally found in the liver, we should convince ourselves of an enlargement of this organ, or of the presence of nodules therein, and should also palpate the entire intestines and the mesentery, in so far as they are susceptible to the palpating finger; above all we must not fail to ascertain whether or not free fluid is present in the abdominal cavity.

Finally, we must palpate for a moment the left *supraclavicular region* for the possible presence of a so-called Virchow's gland. The occurrence of such glands, sometimes ranging in size from that of a hazelnut to that of a hen's egg, according to the opinion of most authors (v. Leube, Ewald, Riegel, Rosenheim, Mathieu, Hayem and Lion, Hemmeter, Einhorn, Fleiner) with whom I coincide, belongs to the greatest rarities. In regard to the fact that enlargement of the supraclavicular glands (Tarchetti and others) unquestionably belongs to a late period of carcinomatous development, complete unanimity does not exist even among the few authors who ascribe great importance to this symptom.

That enlargement of the supraclavicular glands is not peculiar to gastric carcinoma alone, but to all intestinal carcinomata, need hardly be mentioned. The condition may throw light upon the cases in which the clinical signs of an intestinal carcinoma are obscure. Such instances have been reported in literature, and every one of experience will remember similar occurrences. Under these circumstances, glandular enlargement of the supraclavicular region may aid in the diagnosis.

The same is true of another variety of glandular enlargement which has been observed for a long time, especially in France and also in Germany, but in the main has received but little consideration: The enlargement of the *peri-* and *para-umbilical glands.* Simultaneously, but even alone, the umbilicus may also show metastasis,[1] so that we then speak of umbilical carcinoma. This complication, which Quenu and Longuet[2] a few years ago made the subject of a comprehensive study, is exceedingly rare. Nevertheless, in the course of years I have seen about half a dozen of these metastases. That which has been mentioned as clinically important in enlargement of the supraclavicular glands is, *cœteris paribus,* also true of carcinoma of the umbilicus and its surroundings.

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[1] Cases of primary carcinoma of the umbilicus have been described in literature they are, however, exceedingly rare.

[2] *Quenu* et *Longuet,* "Cancer secondaire de l'ombilic." *Revue de Chirurg.,* 189 T. 16, p. 1897.

This exhausts the leading points of the physical examination.

They are sufficient, as we have seen in the case in question, not only for the diagnosis but also, so far as this be possible, to decide whether or not complications are present. From an examination of the vomited material and from its nature a conclusion, although not an exhaustive one, may be drawn in regard to the *gastric functions,* and it will be apparent that in classical cases of carcinoma of the stomach other methods of examination, particularly an examination of the gastric contents, in a strict sense, are superfluous.

In my opinion these are only necessary when other methods yield insufficient or contradictory results.

As this is often the case, the examination of the gastric contents will be included with the physical examination of the patient in question, although not for the purpose of a strict indication, but for didactic reasons. The patient was prepared for this by withholding food from about eight o'clock of the preceding evening to the following morning.

This method of procedure, i. e., the examination of the empty stomach, is very valuable in daily practice, as by this means without further preparation not only the secretory, but, above all, the motor activity of the stomach may be investigated. In the majority of cases, if the stomach proves to be empty, we may at once administer a trial breakfast and an hour after its introduction the gastric contents may be obtained.

In the case in question the trial breakfast is unnecessary, as the patient's stomach, even early in the morning, contains about one-half liter of residue.

We must first examine the residue carefully.

It consists of a grayish, thin fluid, a mucus-containing mass, as may be noted by pouring the material from one vessel into another, and has an acid but not disagreeable odor. If a portion of the fluid be poured into a test-tube there is no obvious change except that the solid constituents fall to the bottom, but true gas formation which occurs so often in other cases of stagnated gastric contents is not here visible. If the attempt be made to observe gas formation in a ferment tube, no actual production of gas is noted.

Occasionally in the gastric contents or in the vomited material one or several particles of the tumor may be admixed, and if microscopic examination reveals characteristic signs of an atypical neoplasm the diagnosis is virtually certain. It need hardly be stated that macroscopic examination alone under no circumstances suffices, since particles resembling tumor may upon accurate investigation prove to be fragments of mucous membrane, coagulated blood, etc.

CHEMICAL AND MICROSCOPICAL INVESTIGATION OF TI GASTRIC CONTENTS

With this external investigation we now include the chemical microscopical examination of the gastric contents.

As may be noted from the color reaction (tropaeolin paper, C paper) as well as from the results of the phloroglucin, vanillin and res tests, *free hydrochloric acid is not present in the gastric contents.* It however, found that litmus paper was decidedly reddened. The gε contents must contain either much combined hydrochloric acid or salts or organic acids, or all of these combined are found. Of thes are chiefly interested in *lactic acid,* especially as its presence may be easily determined. In fact, as was demonstrated by the iron chlorid lactic acid was present in large amounts. Besides lactic acid, other vo fatty acids are probably present, but their exact determination is dif and they are of little practical importance.

On the other hand, further to determine the secretory insufficien the stomach, the proof that *enzymes,* that is, proenzymes of the g contents, pepsin and pepsinogen, as well as lab-ferment and lab-zyn are absent is necessary. As was shown by the tests which were mad previously mentioned enzymes are absent from the gastric contents (patient in question.

We conclude the chemical examination by testing a portion (gastric contents for blood, using the guaiac test modified by Webe

Test for Blood.—As this test has been but little employed in pra(shall at this point briefly describe it. About 10 c.c. of unfiltered ; contents are mixed in a test-tube with about one-third as much of acetic acid, and the mixture is repeatedly shaken. We then add to same quantity of sulphuric ether, after which we shake it, and quently note whether the mixture shows any change in color. If hemoglobin be present in the gastric contents the ether soon chaı a yellowish-brown color somewhat like diluted Tokay wine.

This yellowish-brown discoloration strongly indicates the pres(hematin. The ether is carefully decanted, and ten drops of guai; ture *freshly prepared each time* and thirty drops of old, strong turpentine are added. If hematin is present, upon long standing a grayish and later a decidedly marked violet color develops in the n

We add to the mixture a few cubic centimeters of distilled wa from ten to twenty drops of *chloroform.* This rapidly takes up t coloring matter, and the reaction soon becomes markedly distinct. the intensity of the blue or violet discoloration we may draw con as to the amount of dissolved hematin present in the gastric cont

The test made in the case of our patient revealed an extraor profuse amount of hematin in the gastric contents.

Finally, a *microscopic* examination of the gastric contents should be undertaken. Under the microscope numerous immotile rods, partly isolated, partly in clumps or forming angles, were observed; these are *lactic acid bacilli*, which were first described by me, later by Oppler, and accurately studied by Kaufmann and Schlesinger. Furthermore, numerous transverse, striped muscular fibers were noted of which a part were still well retained, now and then starch bodies, also fatty acid crystals and fatty acid needles. Occasionally yeast colonies, isolated or beginning to sprout, are noted, but, on the other hand, the sarcinæ so frequently present in these conditions were absent.

Pus in the gastric contents is a significant finding, and its presence is often revealed by the odor. If the gastric contents are spread out thin upon a black pus basin and carefully examined, as a rule particles of pus will be discovered which may be verified by microscopic examination.

In the preceding I have described the most important macroscopical, chemical and microscopical findings in the gastric contents of the case under discussion. Let us now consider the collective importance of the individual findings.

The least weight is to be attached to the macroscopic investigation; it is true the coffee-ground appearance may strengthen the suspicion of carcinoma, yet it is not absolutely proven. But the previously mentioned particles of tumor tissue unquestionably are in favor of a malignant neoplasm.

More valuable is the chemical finding, and here primarily the absence of free hydrochloric acid and enzymes, the fact of abnormal lactic acid fermentation, and the presence of blood and pus in the gastric contents are significant.

The absence of free hydrochloric acid (which, as is well known, was discovered in Kussmaul's Clinic in the year 1879 by van den Velden), even to-day is looked upon as a relatively certain and decisive sign. It is of very frequent occurrence in gastric carcinoma; it was present in 77.5 per cent. of my cases, and other observers give even higher figures. But gastric carcinoma is not the only affection which shows the absence of hydrochloric acid. This symptom is observed in maladies of an entirely benign character: In chronic gastritis, in amyloid degeneration, in severe forms of anemia, in poisoning by sulphuric and hydrochloric acids, in so-called achylia gastrica, in hypertrophic pyloric stenosis, and in rare cases of ulcer of the stomach; moreover, deficient hydrochloric acid is found in carcinoma of other organs, for example, of the esophagus, of the uterus, of the breast, and of the rectum.

Inversely, free hydrochloric acid is occasionally found even in conspicuous excess in well developed cases of gastric carcinoma. An excess of hydrochloric acid is especially frequent in carcinomatous ulcer, to which we shall refer later. But also in undoubted gastric carcinoma, particularly of the pylorus and at the onset of the disease, we quite often hydrochloric acid.

Of greater decisive importance is the occurrence of large amoun *lactic acid.* Arthur Schiff[1] compiled the figures of Hammersc Strauss, Schlesinger, Kaufmann and Ekehorn, which show that 84.4 cent. of *gastric affections running their course with lactic acid ferm tion are gastric carcinomata.* According to the same compilation, th vestigations of Boas, Hammerschlag, Rosenheim, Lindner, Kuttner, R Strauss, Kaufmann and Schlesinger show that lactic acid occurs i per cent. of all gastric carcinomata. Higher figures were given by Cro that is, 78.5 per cent., and Rütimeyer,[3] 75 to 80 per cent.

On the other hand, it cannot be denied that lactic acid ferment in the stomach occurs in quite a number of non-malignant gastric tions; for example, in hypertrophic pyloric stenosis, gastritis gravis (R heim), in pernicious anemia with gastric atrophy (Ewald), and in rare cases which, however, do not clinically present the picture of ga carcinoma.

As the presence of lactic acid occurs only with stasis of the ga contents and the absence of hydrochloric acid, it is obvious that defici in lactic acid by no means excludes gastric carcinoma.

The positive finding only is of importance, not the negative.

Besides the chemical changes previously mentioned the absenc *pepsin and lab-ferment* is to be emphasized, to which Hammersc Ewald, Kuttner and others attach great value. This, however, al occurs when the presence of lactic acid is combined with the absen hydrochloric acid, so that proof of the absence of enzymes at most confirms the previously mentioned anomalies or complements them

Occult Gastric Hemorrhage.—In my experience some weight m attached to the evidence of *occult gastric hemorrhage.* This may tected in the gastric contents or, if for any reason not thus perce may be demonstrated in the feces by means of the previously men method (page 230).

Since Ewald and Kuttner called attention to the occurrence of slight hemorrhages, I have recently had an opportunity to study thi ject carefully, and in numerous cases of gastric carcinoma have mined that in these diseases occult hemorrhages are exceedingly co Thus, associated with my assistant, Dr. Kochmann, in 67 cases of carcinoma we found 65 hemorrhages, and in the overwhelming m of cases the bleeding was occult.

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[1] *Arthur Schiff,* "Die Diagnose des Magencarcinoms," "Sammelreferat n Arbeiten der Jahren 1894–1898." *Centralbl. f. die Grenzgebiete der Me Chirurgie,* 1898, Nr. 12 and 13.

[2] *Croner, Mittheilungen aus den Grenzgebieten,* 1899. Bd. V, p. 405.

[3] *Rütimeyer, Correspondenzbl. f. Schweizer Aerzte,* 1900, Nr. 21.

However, this finding is not pathognomonic. We have noted the condition in a large series of observations, most numerously in cases of benign dilatation, also in hypertrophic pyloric stenosis and in ulcer of the stomach, but not, however, in positive cases of chronic gastritis and achylia. Naturally, even in these cases, fresh visible hemorrhages may appear as capillary hemorrhages which, however, as a rule, are of artificial origin. Continuous hemorrhage in chronic achylia or gastritis without conditions of stasis belongs to the greatest exceptions; in carcinoma, however, it forms the rule.

The differentiation of a benign from a malignant pyloric stenosis by demonstrating the presence of blood is not readily made. But we may state that under these conditions the reaction in carcinoma is decidedly more marked (saturated blue) than in benign processes (delicate violet).

Microscopic examination has a certain importance inasmuch as it reveals the thread bacilli of lactic acid fermentation, and thus confirms the presence of the latter. *Sarcinæ*, in my experience, are of no diagnostic significance in gastric carcinoma. They are absent, as a rule, but not invariably, in marked lactic acid fermentation, and, *vice versa*, with the finding of free hydrochloric acid and marked stagnation they are almost always present in carcinoma.

Pus in the gastric contents is by no means a rare symptom; usually it is found admixed with blood. Although pus may occur in the stomach from various causes (gastritis phlegmonosa, abscesses) and is especially prone to originate from the upper air passages, the continuous finding of amounts of pus, particularly if the conditions mentioned may be excluded, is a valuable sign of carcinoma.

Besides the gastric contents, the composition of the blood in gastric carcinoma has received considerable attention; in the main, however, the results are unsatisfactory. We often find, but not constantly, a marked diminution of hemoglobin and of the erythrocytes. The opinion promulgated a few years ago by Schneyer[1] that there is an *absence of digestive leukocytosis* in gastric carcinoma was found to be incorrect after further examination (by Hassmann, Hoffmann, Sayler and Taylor, Chadbourne, Dolmatow, Marchetti, Rencki and others).

From this description it follows that absolute proof of the presence of a gastric carcinoma, aside, perhaps, from the very rare finding of particles of the tumor which is only possible in well advanced cases, cannot be obtained from the investigation of the gastric contents. Only most careful consideration of all the clinical signs, among them the physical, particularly the tumor, can protect us from an erroneous diagnosis.

With this we reach the important and difficult chapter of the diagnosis of gastric carcinoma.

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[1] *Schneyer, Internationale klin. Rundschau,* 1894.

GASTRIC CARCINOMA

DIAGNOSIS

So long as we are dealing with cases similar to the one just desc the diagnosis of gastric carcinoma is one of the easiest in the rea internal medicine. Difficulties beset us only when the onset is indi and the course atypical, when the symptoms differ in important from those just described, when important objective factors are absent or indistinctly developed.

We may differentiate two categories of atypical development: those in which a tumor in the stomach is not palpable at the mom examination; secondly, if the tumor be present, it does not have the acteristic properties of a gastric tumor, and particularly of a mali gastric tumor.

We shall now concern ourselves with the first class of cases. observation gives us many examples of this kind.

We see, for instance, a patient who for a short time has comp of dyspeptic symptoms: Gastric oppression, anorexia, now and then ing pains in the epigastrium or in the fundus, occasionally also i back, eructations, emaciation, a feeling of diminished dynamic ac etc. Palpation may perhaps reveal slight sensitiveness to pressur other than this no objective signs are observed.

The suspicion of a pernicious disease is aroused, but we may a dealing with a severe form of chronic gastritis or even with one of not infrequent forms of nervous dyspepsia which in the course of lead to an extreme degree of marasmus, and thus very closely sin the picture of carcinomatous cachexia.

In another series of carcinomata we observe an entirely different c The disease begins suddenly as a severe gastritis, or like an acute dilatation: In the midst of health the patient begins to vomit, or, a more or less obvious cause, once or several times large or small an of ingesta are ejected; but notwithstanding careful regulation of tl and the use of all remedies calculated to ameliorate the conditio symptoms do not yield but even increase. The patient permanentl his appetite, cannot take even the most carefully prepared food, an few weeks shows a threatening state of inanition.

Or after indistinct prodromes the patient is suddenly attacke profuse hematemesis or melena. We first think of a latent ulcer stitute appropriate treatment; the hemorrhage ceases but the patie not recover his health, he rejects his food, repeatedly vomits, sor food but occasionally also fresh blood, and the case begins to as serious aspect.

In other cases, the patient shows indistinct dyspeptic sympto fever which is not characteristic, nor does it conform to any of t known types. We think of cryptogenetic pyemia or an irregular attack of enteric fever, of malaria, of tuberculous peritonitis, etc., and finally section shows the presence of a gastric or intestinal carcinoma which was insusceptible to palpation.

This shows but a few forms under which carcinoma may be masked. If we undertook to enumerate them all we should lose ourselves in a chaos of conditions which could not be examined.

Somewhat less intricate but still obscure is the second category of cases in which a tumor is present but in which doubts arise as to whether it belongs to the stomach, or in which the history or malignancy is uncertain. We mention, for example, the difficult differentiation of an old ulcer with thickened borders from a carcinoma or a carcinomatous ulcer, furthermore, the recognition of a callous, indurated perigastritis, a hypertrophic thickening of the pylorus, a hard gall-bladder filled with stones, a gastrolith, a lipoma, a papilloma or myoma, not to speak of sarcoma of the stomach.

In these and numerous other cases which cannot be mentioned here all methods of diagnosis, particularly the examination of the gastric contents, must be utilized, and, as I must admit, frequently without success, to clear up these doubtful conditions. But, nevertheless, cases remain in which nothing but the further course of the disease, and often not even this, but only laparotomy or the autopsy, will decide the question.

To proceed, we must mention some other methods which now and then are employed in complementing the diagnosis.

First among these is the examination of the patient under *anesthesia*. By this means doubtful tumors whose presence in the various abdominal organs cannot be determined with certainty may be better recognized or localized. However, in consequence of more thorough technic in palpation, examination under an anesthetic has in the last few years fallen into disuse. It is best reserved for the cases in which active tension of the abdominal walls from marked meteorism, etc., makes it impossible to obtain a clear idea of the condition of the viscera.

The second method is *gastroscopy* which, however, up to the present time has secured no permanent place in the diagnosis of gastric pathology. Invented by v. Mikulicz, and further developed by Rosenheim, Kuttner and Rewidzoff, this method has been chiefly employed in the last few years by G. Kelling. Kelling is of the opinion that gastroscopy enables us to make the diagnosis in doubtful cases of gastric carcinoma, and in other instances to make it early (see below). We must wait and see what the technic and possibilities of gastroscopy achieve in the future, before we can look upon this method as positive and decisive.

So-called *exploratory laparotomy* has also been employed as a diagnostic aid. As a matter of fact every laparotomy is an exploratory incision, as only this gives us accurate knowledge as to the seat of the tumor, its physical composition, its relation to neighboring organs, the existence complications, etc.

In this respect, exploratory incision is in truth the best and most tain diagnostic method. But this opinion is not generally held nor the recognized, above all among the laity. Although with the great deve ment in technic as well as in antisepsis and asepsis the risk is not gr nevertheless the method is rejected even at the cost of a positive diagn In these instances laparotomy is frequently begun as a therapeutic m ure and ended as an exploratory operation.

I believe an exploratory incision to be unjustifiable *when the diagn is wholly obscure or when we entertain the suspicion of a malign process but cannot possibly determine the organ affected.* Here fro simple exploratory incision a lengthy, severe, and occasionally ineffec operation may result. In describing the indications for the opera treatment of gastric carcinoma we shall again revert to this subject.

From what has just been stated the impression will be given tha the majority of cases of gastric carcinoma a clear diagnosis can be m Much more perplexing is it to determine at the onset with certaint probability the exact seat, the extent of the process, the presence of me tasis, a beginning ascites or other complications. In so far as we are d ing with objectively demonstrable changes a careful examination will n rally assist us. Other very frequent occurrences, such as metastasi the liver, or of the mesentery, etc., sometimes escape recognition. *A d nosis entirely comprehensive in this respect is impossible.*

Intimately related to these difficulties is the much discussed ques of the *early diagnosis of gastric carcinoma.* Even the prior ques "What do we understand by early diagnosis?" is not easily answ It has been said "that a diagnosis is made early if no palpable tum present." This is generally true. But the facts show that even in roth's time radical operation was attempted for gastric carcinoma, proves that this requirement was not absolutely a prior conditio successful surgical treatment but also, inversely, what every one o perience knows, that the impossibility of feeling a tumor is no pro conditions favorable to operation. Neither will the time which has el since the onset of the disease furnish us any clue to the distribution process, as groups of tumors which grow rapidly and also such as very slowly have been observed.

The chemical findings, particularly the triad, stagnation, abse hydrochloric acid, and presence of lactic acid, will in some of the vidual cases enable us to make an early diagnosis even although the elude discovery, although isolatedly there are benign processes wi same chemical finding, and, notwithstanding the fact that the p of this triad of symptoms indicates conditions already very unfa for the removal of the tumor.

Nevertheless, in the points above mentioned, collectively at least, and so long as no better criteria exist, these factors in a large number of cases permit an early diagnosis.

The usually very decided period of latency forms the greatest obstacle in the discrimination of gastric carcinoma. In this stage the patients have but relatively few symptoms, do not consult a physician, and resort to home remedies. Certainly most of the carcinomata which we meet are already well advanced and manifest conspicuous symptoms, above all, a palpable tumor. In this condition gastroscopy—even should the technic become more simple—will not materially aid us. Nothing is easier than the diagnosis of rectal, uterine or mammary carcinomata, and yet in these we hear of precisely the same perplexities on the part of the surgeons and gynecologists as in the case of gastric carcinoma.

Nevertheless it would be a great mistake to allow these obstacles to daunt us, and to relinquish our endeavors to make an early diagnosis. The marked successes reported in literature resulting from early operation after an early diagnosis of gastric carcinoma should encourage us, even with the basis of our present methods, always to attempt to diagnosticate the disease as soon as possible.

A special form of carcinoma still to be described is *carcinomatous ulcer.* Previously investigated by Rokitansky and Dittrich, carcinomatous ulcer has recently been thoroughly studied by Hauser. Opinions as to the frequency of the transformation of a simple ulcer into carcinoma are to-day widely asunder. According to Lebert, 9 per cent. of all gastric cancers are due to this cause. Rosenheim found it only in 5 to 6 per cent., and Ewald has lately declared carcinomatous ulcer to be far more frequent than has up to this time been assumed.

According to my experience a positive diagnosis of carcinomatous ulcer *intra vitam* is impossible. The chemical examination of the gastric contents utterly fails us. The excess of hydrochloric acid which Rosenheim previously believed to be decisive is found, as we now know, also in carcinoma without ulcer. I have repeatedly observed in carcinomatous ulcer severe, rapidly succeeding gastric hemorrhages. Yet even these are not decisive. We should always think of *carcinoma ex ulcere* if positive symptoms of ulcer have for a long time preceded the process.

COURSE

The course of gastric carcinoma depends upon various factors, primarily upon the nature and seat of the tumor. Experience has shown that there is a gliding scale for the malignancy of carcinoma; that, for example, adenocarcinomata grow more slowly and less frequently develop metastasis than the rapidly growing, speedily decomposing medullary carcinomata which readily promote the carrying of carcinomatous products.

Scirrhus is again characterized by its tendency to advance rapidly cause stenosis, colloid carcinoma by its unlimited growth.

Possibly *the seat* of gastric carcinoma is of even greater significa in the course. It was formerly assumed that the point of predilec was the pyloric region. More recent experience, as well as operations autopsies, shows this to be no longer true. Carcinomata of the sma curvature are not less frequent than those of the pylorus, and it is e likely that, similar to the course in ulcer, the smaller curvature is preferred seat for carcinoma.

No matter how this may be, the fact is generally accepted that nearer the carcinoma to the pylorus the more rapid and more seri the course of the disease. Under these circumstances the malady set in with elementary force and in an exceedingly short time, eve few weeks, cause death (*acute carcinoma*). In opposition to this are slowly growing carcinomata with numerous variations which are clinic ill-defined, and, according to whether improvement or aggravation app keep the patient and the physician wavering between hope and fear.

The cause of this latency is easily recognized if we remember tha carcinomata not situated in the pylorus vomiting is absent or only pre to a slight extent, that hemorrhages also are but moderate, and t finally, the ingestion of food and its assimilation are far less dama than in the first mentioned group.

Under such circumstances the patients may for a time gain in wei and subjective activity; nevertheless, the outcome shows that the physi was correct in his grave prognosis.

It need scarcely be mentioned that the course of the pathologic pro is influenced to a decided extent by the most varied complications, as ascites, proliferation into the peritoneum, external rupture or rup into the intestine, metastatic pleurisy, etc.

We see from this that in a given case the determination of the dur of the course is beset with great difficulty. However, it may be estim on the average at a year to a year and a half. If this period has ela we will rarely go astray in excluding a malignant process.

COMPLICATIONS AND SEQUELS

The complications which may occur in the course of gastric carci are numerous. Some of these are due principally to the nature o affection, and for this reason are not of great clinical interest. O however, require a brief description.

I must first call attention to the *fever* which occurs in carci this was mentioned by Wunderlich in his well-known text-book, an been more accurately described by Hampeln (1884).

Carcinomatous Fever.—According to Freudweiler, who made a ough study of carcinomatous fever in 475 cases of carcinoma in the Zurich Clinic, this symptom was present in 117 cases (i. e., in 24.6 per cent.), and was not due to complications; rarely (that is, in 1.5 per cent. of all cases) the fever was almost continuous, usually it was remittent or intermittent (in 10 per cent. of the total number of cases); much more rarely it was typically intermittent and resembled malaria (in 3.6 per cent. of all cases, in 14.5 per cent. of febrile carcinoma); in the latter case, the fever was not so typical as in malaria, often it was of an inverse type, the time of its appearance varying on different days as did the duration of the febrile period; in a number of cases the temperature rose but gradually instead of suddenly as in malaria. With relative frequency (in 38.5 per cent. of the fever cases and in 9.3 per cent. of all cases of carcinoma) there were isolated rises in temperature which did not last longer than three days.

In the majority of the patients the condition was that of ulcerative carcinoma so that the fever was attributed to secondary infection; in the minority, however, no ulceration could be detected at the autopsy. Therefore, carcinoma in itself, similar to tuberculosis and lympho-sarcoma, has pyrogenetic properties.

Inversely, as Freudweiler has shown, carcinoma may also run its course with subnormal temperature. In 47.8 per cent. of his cases temperatures below 96.8° F. were repeatedly found.

Coma Carcinomatosum.—A second important complication is *coma carcinomatosum* which was first described by v. Jaksch, then by Senator, Riess, G. Klemperer and others. *Acetone* was first found by v. Jaksch in the distillate from the urine, and Klemperer later detected *oxybutyric acid.* Coma carcinomatosum resembles diabetic coma except that, in my experience, its course is not so rapid. In one of my patients with rectal carcinoma, in whom a few months previously colostomy had been performed, the comatose condition lasted very nearly two months. Whether in these cases, as is exceedingly likely in diabetes, there is an acid intoxication, or whether specific toxins generated by the carcinomatous poison are operative, is not yet known.

Tetany is very much more rare than the two complications just mentioned. This form of tetany does not differ from that occurring in benign pyloric stenosis.

In conclusion we must mention the dropsical symptoms in carcinoma (edema and ascites), venous thrombosis and metastases in the liver which, according to Lebert, occur in 50 per cent. of all the cases, also metastases of the pleura and of the lungs, and finally the appearance of multiple neuritis.

PROGNOSIS

The prognosis, as I need hardly mention, is very unfavorable provided surgical intervention does not soon interrupt the course for a time (unfortunately, however, not for a very long time). As we shall show in the chapter upon therapy, internal treatment may bring about amelioration of many of the symptoms and increase the strength, but the unfavorable outcome cannot be averted. When no complications are present, the prognosis depends mainly upon the nature and seat of the tumor. As I have already mentioned, carcinomata which do not implicate the pylorus run a much slower course, I might almost say a more benign course, than carcinomata which cause stenosis. As regards danger to life, such carcinomata may be prognostically designated as more favorable, but with the limitation that under a radical operation the chances of success are even less than in those of the pylorus.

CARCINOMA OF THE INTESTINES

Proceeding downward from the stomach we meet with carcinomata everywhere but—and this is of great importance—by no means with the same frequency. While carcinomata of the small intestines are extremely rare, carcinomata of the rectum are about as numerous as those of the esophagus. Between these, carcinomata of the large intestine are found; and here also the curvatures are the points of predilection. From this it is evident that, a fact to which Virchow called our attention, those portions of the digestive canal through which the contents move but slowly and sluggishly are particularly predisposed to the development of malignant tumors.

SYMPTOMS

I shall first describe a typical case of carcinoma of the large intestine, then one of the rectum, and in connection with these shall briefly delineate the symptomatology of carcinoma of the small intestine.

The patient was a man in the forties and, except for the diseases of childhood and other mild affections, he had up to the present time always been well. In particular he had never complained of gastrointestinal disturbance. Here we observe the abrupt onset in the midst of perfect health to which I called attention when describing carcinoma of the stomach.

The patient stated that the first symptom was stubborn constipation, so that at the onset of the affection he was compelled to resort to active purgatives to regulate the function of his bowels. Following the constipation attacks of severe colic soon appeared, first at long intervals, and later every two or three days. Finally, he complained of marked loss of weight, lassitude and inability to follow his occupation.

It is evident here that the onset of the affection was but slightly characteristic.

At first a mild coprostasis, a flatulent colic appears, such as we are accustomed to see every day. But, under these circumstances, it is well not to be too optimistic, even in cases in which there is no serious indication of a malignant affection, particularly when an obvious loss of weight cannot be attributed to alimentary processes.

In every case it is well to bear the patient in mind. Repeated weighings should be made, and the physician should not be content until he has decisive evidence whether a mild, or, *vice versa,* a very serious malady is present. Too great pessimism will do no harm, especially if it should later give way to positive optimism.

So much as to the onset of the disease. Let us now discuss its further development. In the last few weeks the intestinal cramp increased to an unbearable degree. The patient was compelled to take narcotics. Purgatives which at first had been active were now ineffectual even in large doses. Stronger drugs were all the time taken, and finally drastics were employed, without relief. The feces no longer appeared in solid, cylindrical masses as formerly, but in the shape of small scybala, frequently admixed with mucus or as a small quantity of thin fluid, or, finally, in this or that form. The patient has not noticed blood in the dejecta. Upon being questioned he reports that he frequently noted simultaneously with the attacks of pain, but often also without these, a distinct, intestinal rumbling similar to the dripping of a fluid, and that this was even audible at some distance. Very observant patients will occasionally state, upon being asked, that occasionally, especially if active pains are present, the prominence of some portions of the intestines has been perceived.

Although these symptoms are by no means positive signs of the nature of the affection, nevertheless it is wise not to place too low an estimate upon them. I can positively state that, from a history of this kind all the details of which have been taken into consideration, I have made a diagnosis of carcinoma of the large intestine even before the patient undressed himself for a physical examination.

Absolute certainty, above all, is furnished by the clinical findings. I shall not allude to the general symptoms, which were discussed in the previous chapter on visceral carcinoma, but shall only consider what may be demonstrated locally.

In intestinal carcinoma the finding of a well-characterized *tumor* is unquestionably the most positive and decisive symptom.

Let us discuss the local findings in the patient in question. As was readily observed, there was an apparently hard but slightly movable tumor about the size of a mandarin in the cecal region, and this could be palpated.

Does the tumor belong to the intestine, and to which part? Are we dealing with a malignant tumor? What is its degree of movability? As these questions arise in every case that is not absolutely clear, their immediate investigation is imperative.

The fact that the tumor belongs to the intestinal canal may, as a rule, be determined without great difficulty from the history in connection with the other clinical findings. Nevertheless, sometimes perplexities arise which greatly annoy the diagnostician. A displaced lobe of the liver, hepatoptosis, with a gall-bladder full of stones or showing carcinomatous degeneration or dropsy, normal but low-lying kidneys, renal tumors, tumors of the omentum, and even tumors of the pylorus may, under some circumstances, simulate neoplasms of the large intestine. In the chapter devoted to diagnosis I shall enter upon these details more minutely.

To prove whether a benign or a malignant process is present is by no means always easy. Chronic perityphlitis with decided induration, tuberculous thickening of the cecum, not to speak of other benign tumors (fibromata, lipomata, myomata, etc.), may resemble malignant neoplasms to such an extent that the true condition is recognized only after prolonged observation, often only at the operation or at the post mortem.

In regard to movability, the law generally obtains that in intestinal tumors this should exist to a high degree. This general rule does not coincide with my experience. I can only admit that the most extreme grades of movability are observed in carcinomata of the large intestine; yet, when a great number of cases are studied, we find only slight, passive locomotion to be present. In the development of the tumor adhesions to neighboring organs very soon form.

For distinct passive movability in inflation of the rectum with gas we have a method which is usually serviceable, but the intestines must not be too greatly distended with gas for, if this be the case, the tumor is covered by the intestines and is palpated with great difficulty.

In the case in question it was readily determined that the tumor was hardly movable, and I may add that upon repeated investigation its position was unchanged. The inflation of the intestines per rectum gave no other results.

Where there is undoubtedly a hard tumor of the large intestine, as well as other points in the clinical history, the clinical picture of carcinoma of the large intestine is sufficiently characteristic; there can be no doubt of the diagnosis. Other factors which we shall later consider at most serve to complement the pathologic picture.

Very frequently, however, the symptoms are complicated. A tumor is absent. There are signs of a malignant intestinal affection but without positive indications as to its nature. Frequently we note a symptom which we also find in the case under consideration: The picture of *chronic intestinal stenosis.* As we have in this volume a masterly description by Nothnagel of chronic intestinal stenosis (which see) I limit myself to a few salient points which relate particularly to carcinoma of the intestine.

Intestinal Rigidity.—In this condition the severe *attacks of obstruction* accompanied by severe colic, occasionally by vomiting, which have already been described are subjectively decisive. Objectively, we must consider meteorism, suprastenotic succussion, and the visible, tetanic, intestinal contractions to which Nothnagel has given the appropriate name of *" intestinal rigidity."*

In well-developed cases of intestinal stenosis meteorism is rarely absent, but is present in varying degree according to the individual case. Sometimes it is localized, and permits a somewhat accurate recognition of the seat of the intestinal stenosis, or it may be general in the stage of intestinal paralysis, that is, of threatening ileus, and may then be indistinct or obliterated.

Intestinal rigidity is the clearest and the most objective symptom of intestinal stenosis. In well-developed cases we note from time to time that the coils of the intestine above the stenosed area contract tetanically with sensations of actual pain, they become hard and stiff, and remain in this condition for some time. With a distinctly audible murmur that I have designated a pressure murmur, this contraction suddenly relaxes, or to a certain extent remains permanently. These are the classical types of intestinal rigidity, but there are numerous deviations. The intestine may contract, for example, but not tetanically, it may become hard and only show an increase of tonicity similar to what we have observed in the stomach, or it rises slightly in the abdominal wall and rapidly sinks without producing acoustic phenomena in its state of relaxation.

The extent of the stiffened intestinal areas varies greatly, according to the seat and the extent of the stenosis. Where stenosis is very marked and provided the seat is in the cecum or in the ascending colon, although rarely when deeper, we note how the small intestines rise one above another like the pipes of an organ, covering the entire abdominal surface, as has been graphically described by Nothnagel.

It is scarcely possible to confound this intestinal rigidity with the indistinct peristaltic motion which we meet with physiologically in well-developed diastasis of the recti, or in motility neurosis of the intestine (tormina ventric. nervosa, Kussmaul). The absence of pain, of rigidity, of constipation, of intestinal splashing, of a pressure murmur, indicates without further consideration the marked difference.

Although intestinal rigidity points only to the existence of stenosis, and only exceptionally to its seat, we are frequently able to make a diagnosis of intestinal carcinoma as well as from the remaining clinical course even in the absence of a tumor. Naturally, this is not always an early diagnosis but, under some circumstances, the conditions may be so favorable as to permit a radical operation which may bring relief to the patient for a long time.

In another group of intestinal carcinomata we find no symptoms of intestinal stenosis; these are the cases which from the onset show no annular band formation with cicatrization, but in which diffuse, infiltrating, ulcerating tumors are found, as in the curvatures of the stomach.

The symptoms in these cases—provided a well-developed localized tumor be absent—vary greatly, according to the stage, the seat, and the extent of the process. It may happen that for weeks and even for months positive intestinal symptoms are entirely lacking. Or, if they are present, they point to a severe intestinal affection the nature of which for a long time cannot be discerned.

In such cases periodic, persistent *investigation of the feces* may in the course of time show the true nature of the condition.

The investigation of the feces in intestinal carcinoma has not the same clinical importance as the examination of the gastric contents in the diagnosis of gastric carcinoma. But a few factors are, nevertheless, significant. Among these is the single or repeated admixture of blood with the feces. This may introduce intestinal carcinoma, or may appear in its course, or may form the terminal symptom.

The hemorrhages may be copious, or may appear as small specks which are hardly visible, or, finally, they may be occult and only detected by chemical examination (guaiac test) or by the spectroscope. Kochmann and I observed such occult hemorrhages in two cases of carcinoma of the colon, and greater attention will, therefore, subsequently be devoted to this symptom.

In the main, however, copious intestinal hemorrhages are not common symptoms of intestinal carcinoma. Such an experienced investigator as Treves[1] calculates their frequency at only 15 per cent. Whether, however, smaller hemorrhages which readily escape recognition by the patient and the physician are not more frequent is yet to be determined.

Besides pure hemorrhages, admixtures of *blood and pus* have been observed by other authors as well as by myself. I have repeatedly noticed these in deep-seated intestinal carcinomata (in the sigmoid flexure) which offer more favorable conditions for the retention of pus corpuscles than, for instance, the cecum or even the small intestine.

That mucus also is sometimes admixed with the feces is readily conceivable if we remember that the carcinomatous neoplasm produces in a large portion of the intestinal tube a condition of catarrhal inflammation. In a case of carcinoma of the ascending colon near the cecum, determined at the autopsy, I observed even a well-developed membranous colitis.

In regard to the form of the feces, it has previously been briefly stated that various deviations from the normal may be observed.

Thin, flattened, tape-like feces have been designated as typical of stenosis. We know to-day that this is untrue, not because normal feces may temporarily appear in stenosis nor because the changes mentioned indicate the formation of stenosis. Only one condition of the feces is never found in carcinoma causing stenosis, namely, a *persistently well-formed, cylindrical shape.*

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[1] *Treves*, "Intestinal Obstruction," London, 1899.

In very isolated cases of intestinal carcinoma, just as in gastric carcinoma, shreds of the tumor are found in the feces. The few observations at hand do not permit a conclusion as to the clinical value of this symptom; nevertheless, this much is certain, that sequestration only occurs in well advanced cases.

In this description I have considered mainly the high-seated carcinomata of the large intestine. The deeply-seated ones, therefore those from the flexura colico-lienalis downward, are marked by certain peculiarities which I must briefly indicate.

Here *tenesmus,* which we must consider in detail under the description of carcinomata of the rectum, becomes prominent, not only tenesmus recti alone but combined with tenesmus vesicæ.

Rectal Carcinomata.—Occasionally the latter may be so prominent that we are inclined to think of an affection of the bladder. If a tumor be present it is situated either in the region of the sigmoid flexure or closely adjacent to the bladder where it may be felt below or beside this organ. In one of my cases the tumor was situated at the right side of the bladder, and was therefore thought to be a cecal tumor. Occasionally, however, carcinomata of the sigmoid flexure, as Körte correctly remarks, are not susceptible to palpation, that is, when their situation is intermediate, or just above the rectum. They cannot be reached by digital examination either from above or below, and offer great difficulty in diagnosis as well as in surgical operations. Carcinomata of the sigmoid flexure lead immediately to the description of the most deep-seated intestinal carcinomata, those of the *rectum.* We shall proceed to consider these by quoting a concrete case.

The patient, as was at once apparent, showed no cachexia, nevertheless the history denoted that we were dealing with a serious affection. He was 52 years of age. He stated that his mother perished after several operations for a tumor of the uterus, his father died from apoplexy. The patient gave an excellent history of himself up to the time of the present disease, which began about five months ago with a slight sensation of pressure and inability to evacuate the bowels naturally. The feces were very frequently admixed with mucus, gradually tenesmus became more marked, and every three or four hours the patient was compelled to seek the toilet, frequently without other result than thin fluid feces admixed with mucus, and even then showing traces of blood.

Thinking he had intestinal catarrh he resorted to a diet consisting of non-irritating gruels and soups with the result that tenesmus was produced and his strength and weight decidedly decreased.

Among the symptoms tenesmus became prominent and this at once indicated that the seat of the affection was in the rectum. Of course in all cases in which this symptom is mentioned we do not invariably at c think of carcinoma, as the same is complained of by patients with hen rhoids, with acute and chronic proctitis, with ulceration in the rect with prolapse of the rectum, with so-called fecal impaction, and ever purely nervous spasm (proctospasm) this symptom is by no means r *Nevertheless, whenever we have the symptom of tenesmus a careful dig or ocular exploration of the rectum should at once be made.*

Before describing the patient's condition we shall first determine whe physical examination of the thoracic organs and external palpation reve any anomaly. This was not the case except for one suspicious point. found that the liver was decidedly enlarged. It projected beyond border of the ribs about three fingerbreadths, and the border was spicuously hard to the touch but not otherwise painful. Digital exam tion confirmed our suspicion. High up in the rectum a hard, nodula scarcely movable tumor was felt, and, as was observed when the fi was withdrawn covered with blood, an ulcerative tumor.

This made the diagnosis in our case positive. The only question wl remained was whether the enlargement of the liver was connected with carcinoma of the rectum. I believe we must answer this in the affirma as rectal carcinoma is especially prone to cause metastatic processes means of the hemorrhoidal veins.

To complement the examination of the tumor we may view the mo focus directly by means of a speculum which is frequently employed this purpose. However, I regard digital examination as decisive and ally sufficient. The conditions are different with high-situated tu which are difficult or impossible to palpate with the finger. Here, have convinced myself, the diagnosis may sometimes be made by an e ination with the speculum. If this also fails, examination unde anesthetic should never be omitted.

In carcinoma of the rectum in which a tumor can be palpated e *ination of the feces* is of secondary importance. Where the tum located high, and inspection and palpation give no result, careful systematic examination of the feces may guide us to a correct diag from the fact that very frequently or constantly blood, mucus and pus occasionally desquamated shreds of tumor, are found. At all even should not rest until we have satisfactorily explained these very con ous changes.

Various deviations from the condition just described may mislea physician who is not familiar with these symptoms. I can quote from my own rich experience without by any means exhausting In some patients the disease sets in with decided hemorrhage which, l once occurred, is soon repeated. The patient believes that the bleed from a hemorrhoid, and, assisted by popular literature or by his phy is content with his "golden vein." In other cases marked diarr actual tenesmus do not point to the rectum but to higher portions of the intestine. In still other instances I have observed how accidental complications such as minor genital affections (in one case uterine retroflexion) so engrossed the attention of the physician that the affection of the rectum was entirely overlooked.

In three of my cases pregnancy was present, and the patient's symptoms were attributed to this until digital examination revealed the existence of a rectal carcinoma. In two of these, fortunately, extirpation was possible.

Simple as the condition appears, the recognition of a carcinoma of the rectum is difficult when the affection has not developed along normal lines, and when it can only be recognized by careful digital examination, which is necessary in every such case of intestinal disturbance.

Cancer of the Small Intestine.—*In carcinoma of the small intestine* we are on much more uncertain ground. Carcinoma of the duodenum shows such similarity to the clinical picture of gastric carcinoma that a differentiation between them is simply impossible. At all events, in carcinomata situated below the papilla of Vater with the constant regurgitation of bilious masses, we may assume a stenosis in the region of the pars descendens duodeni and not beyond this unless, exceptionally, a clearly localized, easily palpable, and otherwise characteristic tumor points to the nature of the affection.

The recognition of so-called *papillary* carcinomata, i. e., those situated and growing around the papilla, is extremely perplexing. Jaundice develops very rapidly, but it is simply impossible to determine its cause on account of the many conditions which are here present. If a tumor can be palpated, sometimes even without this, the diagnosis of a malignant affection may be more or less accurately made from the course of the disease. The determination of its seat is, however, always accidental. In all carcinomata of the duodenum gastric disturbances are the most prominent, particularly vomiting, while symptoms attributed to the intestine are of a secondary nature.

What is true of papillary carcinomata is, in the main, true of the exceedingly rare carcinomata of the *jejunum* and *ileum*, especially as these only exceptionally lead to the formation of a tumor. When a tumor is present it is distinguished by its great movability. Occasionally, when symptoms of stenosis develop with constant vomiting of fecal or fecaloid masses, we may diagnosticate the seat of the tumor as in the jejunum. The appearance of intestinal hemorrhages may then be a further indication of the nature of the disease. The subjective symptoms, in the main, do not differ from those of deeply situated duodenal carcinomata.

DIAGNOSIS

The diagnosis of intestinal carcinomata, as is evident from the fc going, meets with great and occasionally insurmountable obstacles. shall not refer to these again, nor shall we enumerate all the errors wh may diagnostically come into question, as it is impossible to exhaust list. We hold that the decisive and most important symptom is the find of a tumor in the intestine, and even this, aside from the difficulty of positive localization, does not confer absolute certainty. Thus, for exa ple, a frequent source of error which has lately been much discussed found in inflammatory tumors of the cecum.

Here, primarily, the differentiation of intestinal carcinomata from bercular ulcers comes into question.

Obrastzow[1] mentions the following points in the differential diagn of these conditions: In carcinoma of the cecum the intestines themse cannot be palpated, but the tumor with the cylindrical mass attached t and extending downward as well as the ascending colon passing upwar palpated. In contrast with this, in tuberculosis of the cecum the intes with its characteristic peculiarities is for the most part susceptible palpation, but its walls appear thickened and infiltrated. Moreover, pation usually shows the tumor of carcinoma to have sharply def borders, while in tuberculosis the infiltration more or less gradually appears. Finally, in carcinoma of the cecum stenosis very soon occu

These points may be unquestionably looked upon as auxiliary fac but it is doubtful whether in the majority of cases they enable u reach a positive decision.

The *constant finding of tubercle bacilli* in the feces without si taneous pulmonary tuberculosis, as mentioned by Obrastzow, is far decisive. If this is confirmed we will have an additional diagnostic terion. Besides this, the age, the duration of the disease, the state o lungs and the course of the fever will aid in the diagnosis. Collec in isolated cases they make a definite opinion possible, in others thi be tentative, and will remain so until later clinical signs (ascites, or laparotomy remove all doubts.

Besides cecal tuberculosis other inflammatory processes from the v form appendix, and occasionally also from the typhlon, may assu tumor-like character so that in deciding as to their nature certain arise. Errors in diagnosis are frequently made in surgical cases fro fact that malignant tumor has been assumed where a simple exud present, and *vice versa.* But by careful clinical observation these as a rule, may be avoided.

The same is true of the exceedingly rare cases of benign tumors cecum (myomata, lipomata, fibromata, etc.), while the differentiation from sarcomata is only possible under very favorable clinical circumstances.

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[1] *Obrastzow, Arch. f. Verdauungskrankheiten,* Bd. IV, 1898.

Benign infiltrations may occur in the sigmoid flexure as well as in the cecum (sigmoiditis); occasionally they are of the consistence of a neoplasm and thus make the diagnosis difficult or impossible.

Spastic contractures of the colon must here be referred to; they are frequently associated with obstinate habitual constipation, and are found particularly in the transverse colon, in the cecum, and are occasionally very conspicuous in the descending colon and the sigmoid flexure. As a rule, however, it is possible at once to determine a functional spasm by inflation or by filling the intestine with water. By this means the spasm completely disappears to develop anew after a short time.

We are here upon more positive ground than in the diagnosis of *tumors of the rectum.* In so far as they may be reached by the finger or the speculum, they rarely cause confusion, and above all, *benign neoplasms and syphilitic strictures* here come into question. In regard to the first, with the exception of polypi, which are extremely rare, we observe that benign tumors of the rectum never produce ulceration, while these as a rule occur very early in carcinomata of the rectum; nor does stenosis occur, at least not the very characteristic, rigid stenosis of carcinoma. I must not fail to mention, however, that benign tumors of the rectum, particularly polypi and papillomata, not rarely in later years lay the foundation for carcinoma. I have observed a case of this kind.

Well developed *syphilitic stenoses* present an entirely different appearance from carcinomatous ones. As Kraske[1] correctly states, syphilitic ulcerations never have the coarse, tumorous border of the malignant, and, unlike these, are usually multiple and separated from one another by areas of normal or cicatricially altered mucous membrane. In doubtful cases the diagnosis may also be made by microscopic examination of a small excised particle or a portion that has been detached by the finger.

Diagnosis of Cancer of the Small Intestine.—The diagnosis of carcinoma of the small intestine, even when tumor is present, is most difficult. So long as we are dealing with the upper portion of the duodenum and on account of the identity of the clinical course, the differentiation from gastric carcinoma, as has already been mentioned, is simply accidental. Lower down, particularly in the descending part of the duodenum, the conditions are somewhat more favorable, as the permanent regurgitation of bile is a valuable point in regard to the seat of the tumor. Still further down, however, the clinical symptoms again become indistinct, and only exceptionally permit a well-founded opinion.

The nearer the carcinoma of the small intestine is to the ileo-cecal valve, the more closely does the clinical picture resemble that of carcin of the large intestine, and it becomes impossible positively to diagnosti the condition.

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[1] *Kraske,* "Erfahrungen über den Mastdarmkrebs." *Volkmann's Sammlung klin. Vorträge.* 1883–84, p. 789.

All this is true of intestinal carcinomata with determinable tumor mation. Where this is absent the conditions, as a rule, are still more c plex. Not that the diagnosis under these circumstances is impossi on the contrary, for example, stenosed carcinomata of the large intes even without palpable tumors, may furnish typical cases. In othe at least as far as the determination of a stricture of the large inte and, proceeding from this fact, a reflection of the entire clinical pictu a probable and even a positive diagnosis of carcinoma of the intestine be made. Nevertheless, complicating factors frequently mislead the d nostician, so that great care is necessary.

We waver, however, in our opinion when a palpable tumor and sy toms of stenosis are absent. In this case the clinical impression ca most only indicate the correct direction, and this impression is fur strengthened by certain episodes, for example, the onset of severe he rhage, the discharge of particles of the tumor, the appearance of pal metastases, ascites and edema, perforation into neighboring organs w usually occurs at a very late stage, and is unfavorable for "operat treatment. Examination of the feces may mislead us, especially as finding of blood and pus is susceptible of many explanations, and not permit, for example, a differentiation from ulcerative colitis.

COMPLICATIONS AND SEQUELS

The complications and sequels of carcinoma of the intestine in respects resemble those of carcinoma of the stomach. In one as i other febrile conditions may appear, usually in the advanced stages c affection. Coma carcinomatosum has also been observed in cancer c intestine. Metastasis of the liver, ascites, pleurisy, peritoneal carci external or internal ruptures occur—particularly into the peritoneur bladder and the female genital organs. Other sequels belong to the of casuistics, and therefore do not need especial mention at this pl

The chief complication of stenotic carcinoma requires a thorou scription: *The transition of partial into complete obstruction.* Thi increase slowly and gradually to total occlusion, or it occurs sudde a previously permeable intestine and may astonish the patient as v the physician. The latter may be due to several causes: Either the of the hypertrophied suprastenotic musculature, which has functione ceases suddenly and completely, or in and immediately above the st obstructing fecal plugs or a residue of food have accumulated whic withstanding sufficient muscular power, make propulsion impossil finally, a quantity of stagnating fecal masses may produce volvul

In these cases a fulminant intestinal occlusion occurs with threatening danger which can only be averted by an early operation. *Pseudo-ileus* may, however, also be present. The patient presents the severe picture of absolute intestinal occlusion. Internal treatment is entirely without result but, nevertheless, contrary to our expectations, the intestines may again become open, the patient may pass gas and feces, and is for the moment saved. But only for the moment! For the next or a succeeding obstruction may become complete and, in spite of medical treatment, pursue its fatal course unless surgery again intervene at the proper time.

PROGNOSIS

The prognosis of cancer of the intestine, as need scarcely be reiterated, is in the main just as grave as that of other carcinomata. But, in the opinion of numerous surgeons, the tendency to the formation of metastasis is not so great as in cancer of the stomach, so that operation performed at the right time offers in the main better chances than in the latter affection. And, as we shall point out in the chapter devoted to treatment, the possibility of total extirpation, for example, in carcinoma of the colon and of the rectum, is more frequent than in carcinoma of the stomach; the circumstance also that in carcinoma of the colon and of the rectum the nutrition is relatively but little affected makes the prognosis somewhat more favorable than in carcinoma situated higher up.

TREATMENT OF CARCINOMATA OF THE STOMACH AND OF THE INTESTINES

This combined description of the treatment of carcinomata of the stomach and intestines has not been without a purpose. The fundamental laws of internal as well as of operative treatment present in both such numerous points of contact that a collective presentation is by far the best to answer didactic requirements.

Complete cure of a gastric or intestinal carcinoma is unknown, not even by means of the knife. The latter by radical or palliative procedures may decidedly prolong life, not, however, or at least very exceptionally, up to the normal, probable duration of life of the individual in question; but even with the highly developed surgical technic of to-day and the popularity of operations on the intestinal canal, this prolongation of life occurs only in a small fractional number of the patients. According to the statistics of G. Heimann[1] made in Prussian hospitals in the years 1885 and 1886, 14.4 and 15.9 per cent. respectively of patients with cancer of the stomach were treated by operation, and even in cancer of the rectum in the same years only 45 per cent., at most 50 per cent., of the cases were operated upon. We cannot be far wrong in assuming that the remaining number were already in such a hopeless condition that operation appeared to be useless.

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[1] *G. Heimann, Arch. f. klin. Chirurgie*, Bd. LVII, p. 4.

Therefore, the majority of patients with cancer of the stomach, even to-day, must be treated by internal means and in no inconsiderable number of those operated upon this is necessary after the operation (palliative or radical).

Internal Treatment.—The objects of *internal treatment* depend in general upon the following considerations: As an actual cure is impossible it becomes necessary, just as in other internal conditions (gall-stones, diabetes) to keep the affection latent as long as possible, and to ameliorate or remove the subjective and objective disturbances which arise. This may suffice for a certain time but permanently it is inadequate, and the reasons for this are manifold.

Primarily, we must consider the mechanical disturbances which frequently arise in gastrointestinal carcinoma, then certain toxic products which are produced by the carcinoma, subsequently the hemorrhages which mediately or immediately are caused by the carcinoma, also metastasis, and, finally, the disturbances of metabolism which, even though not wholly, are nevertheless in great part the consequence of carcinoma.

In regard to the latter, the accurate investigations of F. Kraus, Fr. Müller, and Klemperer, to which those of v. Noorden-Gärtig must be added, have given us full information. They have unanimously shown that a number of patients with cancer, notwithstanding plentiful food and a high amount of albumin, permanently lose N. Fr. Müller found that on increasing the administration of food the conditions remained unchanged. The N-excretion was invariably greater than the N-intake. The increase of acetone observed by F. Blumenthal in severe carcinomatous cachexia points to an increased decomposition of albumin. The more these products increase, the more marked and persistent is the loss of strength, and *vice versa.*

It is, therefore, evident that the nourishment of patients with cancer is extremely difficult. In addition to this, the appetite in the course of carcinoma is very frequently, although fortunately not always, decidedly decreased, chiefly so in carcinoma of the stomach and small intestine, and to a somewhat slighter degree in carcinoma of the colon and rectum.

We must always bear in mind the importance of nourishing patients with cancer as plentifully as possible. Our individual points of view must depend upon the position and the nature of the carcinoma, and the stage in which the patient comes under professional observation.

Let us begin with cancer of the stomach. We find the conditions vastly different according to whether the carcinoma runs its course with or without very decided motor disturbance, or, *vice versa,* severe symptoms of stagnation are present; in the latter case, as a rule, vomiting minates the scene. In the former case, a decided limitation of diet not indicated, or to but very slight extent. Albumin, carbohydrates, and at, naturally most carefully prepared, are permissible, and above all milk nd milk preparations in the most varied form and according to the individual circumstances and the wishes of the patient.

With a fairly good appetite and the careful combination of foods, it may be possible to keep the patient for weeks and months in good condition, and now and then even to increase his weight. Occasionally the patients are subjectively better, and their courage revives, a fact to which the gain in weight conduces not a little.

It is unfortunate that most patients with cancer of the stomach have a distinct repugnance to meat and meat derivatives (therefore to bouillon and meat extracts). Hence the administration of meat cannot be insisted upon, but an attempt should be made to administer other albumin bodies, with eggs, carbohydrates rich in albumin, dishes prepared with milk, etc. After a prolonged abstinence from meat an attempt may be made again to administer it in some form, but usually without success.

The nourishment of the patient is much more difficult in carcinoma at or near the pylorus. Here, as a rule, we are limited to fluid food, and ften but small quantities will pass through the pylorus. Above all, milk reparations, soup with the yolk of an egg, the various leguminous vegeables, and cereals in convenient form, may prove serviceable. That only mall quantities should be administered will be understood. From the uantity of urine voided, and by comparing the amount of material found the stomach by lavage in the early morning with the amount of fluid ntroduced, we can practically estimate the amount absorbed.

It is perhaps well at this point to touch upon the value of *artificial ood preparations* in cancer of the stomach. We may to-day regard it s certain that they have no beneficial effect on the nutrition.

Their value consists solely in the fact that the patients who have no ppetite for food, but actual repugnance, regard the intake of these subtances mixed with nutritive preparations as a duty to which they submit s willingly as to that of taking medicine. From this standpoint it is uite immaterial what artificial food we administer to cancer patients. The individual taste will best decide. According to my personal experience the most suitable preparations are: Puro, meat jelly, somatose, sanatogen, roborat. These products will also furnish a sufficient variety.

Luxuries, substances *which tempt and stimulate the appetite* (v. Leyden), are just as important as nutritive preparations. What has been said above is also true of these. By their aid it is often possible for a ong time to keep the patient away from the cliffs of inanition. What is dvisable in the individual case depends upon the social position, upon he habits, and finally upon the wishes of the patient. At all events by eing too strict we may do more harm than good.

Among the auxiliary dietetic measures belong *artificial*, or, as it has lately been called, *extra-buccal* nutrition. For the technic and other details we must refer to von Leube's excellent description. It only remains for us briefly to mention the value of this in carcinoma of the stomach. Unquestionably the mere introduction of fluid to prevent a too dry condition of the organism may also prevent threatening tetany and the accumulation of toxic products, and the intake of even a few calories is a gain which should never be undervalued.

No one will claim that extra-buccal nutrition decidedly prolongs the life of the patient with cancer. The simplest method and the one most in use is by means of *nutritive enemata*; subcutaneous methods (oil-sugar solutions), no matter upon what scientific principle they are based, have, as yet, not found general acceptance.

What is true of cancer of the stomach is also true of *carcinoma of the small intestine.*

In carcinoma of the large intestine other points of view arise, and, above all, it is here an absolute law that we should avoid everything in diet which may obstruct or delay the passage of the feces. *Avoidance of all substances with skins containing residue unsuitable to the gastric and intestinal juices* is therefore a leading principle. Then the nutrition must be calculated directly to stimulate intestinal peristalsis. This may be attained by *physiologic aperients*: Honey, milk sugar, sour milk, stewed fruit, lemonade, wines made from fruit, marmalade, and fruit jellies. Other foods may be given in the form and manner mentioned under the consideration of cancer of the stomach.

In ulcerating carcinomata which do not run their course with the formation of stenosis, we must be particularly careful of the diet. The most suitable articles here are milk and milk preparations, soups made from leguminous vegetables, soups containing eggs, meat jelly, etc. In extreme stenosis from obstruction the introduction of food is most difficult on account of the mechanical hindrance to the passage of feces. Under these circumstances we must limit ourselves to small amounts of highly concentrated food.

The artificial introduction of food in carcinoma of the large intestine is only permissible provided the neoplasm does not extend lower down than the cecum or the flexura colico-hepatica. Below this it will irritate rather than benefit the nutrition. In carcinoma of the sigmoid flexure there can naturally be no question of rectal alimentation. Reports in [illegible] to subcutaneous nutrition, which is here indicated, are not to my [illegible]dge at hand.

[illegible]*ug Treatment.*—To ameliorate the sufferings which carcinomata of [illegible]mach and intestines bring in their train drugs are often useful [illegible]nally even indispensable.

[illegible]e of the most frequent causes of distress in gastric and occasionall also in intestinal carcinoma is *loss of appetite*. Often this can be relieved for a time by the so-called stomachics, by condurango as well as by other bitters; as a rule, however, they fail. In so-called achylia gastrica, which, as before stated, very frequently occurs in gastric cancer, we may employ hydrochloric acid and also pancreatic preparations to improve the digestion. Recently pankreon has been much employed in the form of tablets each containing one gram.

Pain in the region of the stomach and particularly of the intestine also calls for relief. In gastric pain, as a rule, mild narcotics (codein, dionin) suffice. In severe cases of obstruction, in intestinal carcinoma, the most powerful narcotics are necessary. First among these is opium in the form of the tincture of thebain, or pure opium (0.03–0.05) perhaps in combination with belladonna (extract of belladonna 0.01–0.03), then morphin as a powder, in solution, by suppositories or subcutaneously.

Obstinate constipation by no means contraindicates the administration of opium; on the contrary we frequently see that after complete intestinal rest normal intestinal peristalsis again sets in.

Opiates act remarkably well in the severe rectal and vesical tenesmus which we have learned to recognize as regular accompaniments of deep-seated carcinoma. By the influence of these drugs the patients may be spared these distressing symptoms for many hours during the day or during the night.

Purgatives play an important rôle in the treatment of gastric and intestinal carcinomata. In the former purgatives will only rarely be administered, preference being given to lavage. If aperients are indicated, fluids or substances readily soluble in water are preferable to pills and also to tablets which are frequently dissolved with difficulty or not at all.

In the stage of intestinal rest in carcinoma of the intestine, purgatives are usually indispensable, and here also purgatives that are easily soluble yet at the same time are mild and act quickly are the best. As most suitable remedies for this purpose I advise: Fluid extract of cascara, compound licorice powder, castor oil, magnesium, rhubarb, flowers of sulphur and, finally, mineral waters in small doses. In intestinal carcinoma it is wise to keep the bowels regulated by the continuous use of a suitable purgative and thus avoid being surprised by the sudden cessation of peristaltic action.

Mechanical Treatment (*gastric lavage*).—Gastric lavage in cancer of the stomach and small intestine may prove useful and palliative, but even here we must individualize strictly. This should not be resorted to so long as the motor activity of the stomach is unimpaired. It is true that lavage acts favorably upon the pain, also occasionally, when stagnation exists, upon the appetite.

But even when this is the case, the nature of the carcinoma, its stage, and the strength of the patient must be carefully considered. In stag tion not too far advanced lavage may be employed, provided pain, a sen tion of pressure, nausea, vomiting and anorexia are present. Stagnati itself, in my experience, does not necessitate the employment of lava In the course of years I have seen numerous patients with cancer of stomach who, in spite of moderate stagnation, remained in fair conditi for quite a long time.

With severe stagnation and coffee-ground contents I no longer empl gastric lavage, because I have observed that the patients are much wo after this treatment than before. If it be necessary to remove the stagn masses, an expression limited to a few minutes is sufficient. But even incipient cases I believe it wise to resort to gastric lavage as little possible. With care in the diet we are frequently able to prevent abnor collections of material.

Indications for, and Results of, Operative Treatment.—The physici is to-day in duty bound carefully to study the indications for, and results of, abdominal surgery. In advising or rejecting operation, he often held responsible both for what is done and what remains undo He will base his opinion, in the first place, upon statistics of large gro of cases, and then upon personal experience. In any event, no mat how the individual case may present itself, the following questions sure to arise:

1. Is surgical interference indicated in the stage in which this pati presents himself?

2. If this be the case, what operation is indicated, and what are probabilities of success?

In some individuals the answer to the first question is easy, in ot difficult or even impossible. The decision may be quickly made in dea with very cachectic individuals, also in those cases in which diffused ed ascites, diabetes, hepatic metastases, or serious complications on the of the heart, the lungs or the kidneys exist. Under these circumsta naturally, any surgical interference will only hasten the end. Nor we resort to operation if, for example, there is a very circumscribed c noma of the small curvature, which neither subjectively causes s symptoms, nor objectively produces serious motor disturbance. Ex ence teaches us that in such cases the duration of the patient's life less than after operation, provided that exceptionally a radical re is indicated by the position of the growth. According to my expe the prognosis is very serious in those cases of carcinoma (usually a pylorus) in which the vomited material or the evacuated gastric co resemble coffee-grounds. The results which I have seen after even p tive operation can scarcely be called good.

In carcinoma of the large intestine and rectum the conditio quite similar, but the lower down the growth is situated, the more ble are the prospects of a radical operation. We shall revert to this ater.

In regard to the second question, if there are no contra-indications uch as have been mentioned, surgical interference becomes necessary and e must now consider the question: What operation is to be performed, hall it be the radical removal of the tumor or merely palliative treatment, .e., the removal of the motor disturbance?

Let us begin with the *stomach*. In the first place, we must confess hat an absolutely positive decision as to whether extirpation or gastronterostomy is indicated is most difficult to answer *prior* to the laparotomy. o matter how favorable the location of the tumor, laparotomy may show uch a general extension of the carcinoma that a radical removal of the umor is simply impossible. v. Mikulicz and Kausch[1] therefore very roperly remark that every operation is primarily an exploratory laparotmy, and a definite plan of operation depends upon what is found on pening the abdominal cavity.

In many cases we may at once exclude the possibility of total extirpation, for example, when the tumors are large and immovable or when the tumor is movable but the patient has not sufficient strength.

In non-palpable tumors the decision is still less possible prior to exploratory incision. When we have reason to assume that a pyloric carcinoma causes stenosis, a radical operation is at least possible. In other cases, however, even with a positive diagnosis, such an operation can hardly be advised.

Besides gastroenterostomy, as a palliative procedure we should also consider *jejunostomy* which is particularly advised by v. Maydl. He introduced this operation for the cases of gastric disease in which a great portion of the stomach was destroyed, and in which it was impossible to supply sufficient nutrition.

Recently this surgeon has advised us to employ jejunostomy in place of gastroenterostomy, as the former is much more thorough, and, besides, meets the important requirements of the case by sparing the diseased organ and protecting the neoplasm from all irritation.

However, this operation has been employed by surgeons only to a limited extent, chiefly for the reason that, like all operations for fistula, cosmetically it is not fully satisfactory. The prolongation of life, too, is less than after gastroenterostomy.

As a very thorough operation, total extirpation of the stomach, the union of the upper duodenum and the cardia, or the lowest portion of the esophagus, which was first successfully performed by Schlatter in Zurich, comes into question. Although to-day quite a number of favorable results are reported in surgical literature, nevertheless, as v. Mikulicz and Kausch[1] state, metastatic lymph-glands are rarely absent in any case of gastric carcinoma, and, therefore, we should not indulge in too great expectations of the results of this heroic treatment. On the other hand, it must be admitted that some remarkable successes—for instance, the last case operated upon by Fedor Krause—favor the radical removal of the neoplasm. At the present time, however, the definite results, the failures and successes, cannot be positively recorded.

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[1] *v. Mikulicz* und *Kausch*, "Handbuch der praktischen Chirurgie," Separatabruck, p. 147.

In the main, *gastroenterostomy* and *resection of the pylorus* are the operations at this time most frequently performed.

With the results of these methods we are comparatively well acquainted, a large number of statistics being available, as well as the individual reports of eminent surgeons. Thus, Terrier and Hartmann have collected the statistics of abdominal operations performed by distinguished surgeons (Czerny, Krönlein, Carle, v. Mikulicz, Kocher, and Hartmann) and from 127 resections of the pylorus have calculated a mortality of 26 per cent.; but this varies greatly. For example, Lindner has lately estimated the mortality at 50 per cent. In the main, however, the results of resection in the course of the last twenty years have gradually improved. v. Mikulicz[2] computed the total of radical cures at 17 per cent.

If this estimate is compared with the total number of cures of other carcinomata we are by no means hopeless. According to v. Mikulicz, the cases of carcinoma of the breast permanently cured amount to from 10 to 15 per cent., with curettage of the axillary cavity to 25 to 30 per cent., in carcinoma of the uterus 30 to 35 per cent., in carcinoma of the rectum 10 to 20 per cent., in carcinoma of the tongue 10 to 20 per cent. If we compare these with the results of gastroenterostomy, the latter, on account of a more complete technic, and, above all, by the avoidance of the so-called vicious circle, are decidedly better. While, according to the last great statistical report of Chlumky, the mortality from 1881 to 1885 was still 75 per cent., from 1886 to 1890 it decreased to 48.4 per cent., and from 1890 to 1896 declined to 36.61 per cent.; for the past five years v. Mikulicz estimated his operative loss at only 28 per cent. (in his last report v. Mikulicz computes these fatalities at 26 per cent.).

In gastroenterostomy, however, not only the immediate results of the operation but the prolongation of life as well as the functional results must also be considered. As to the former, the average results are not very satisfactory. Life was prolonged for five or six months; now and then there must have been brilliant exceptions to this.

Nor is the functional effect in all cases satisfactory. Frequently the pains and vomiting recur, jaundice, ascites and edema appear, and a persistent anorexia leads to early loss of strength. Even in favorable cases, in spite of an increase in weight, the patients do not gain in strength, and are unable to follow their usual occupations. *In other words, the prolongation of life is frequently nothing more than a prolongation of suffering.* It is true some strikingly favorable results occur, but in the overwhelming majority of cases, after a brief improvement a progressive downward course is noted.

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[1] *L. c.*

[2] *v. Mikulicz, 73. Versammlung deutscher Naturforscher und Aerzte in Hamburg*, 1901. Referat.

It follows from this that, although the immediate results of gastroenterostomy are relatively beneficial, for the duration of life as well as for the general bodily condition and functions, after gastroenterostomy there is still much to be desired.

Under these circumstances surgeons as well as physicians frequently raise the question whether gastroenterostomy should not be limited in favor of total extirpation. After I had formulated this conclusion based upon my own experience,[1] v. Mikulicz, in the article cited above, and from his far greater experience, arrived at the same opinion.

If from this standpoint we make a strict choice, the number of cases suitable for resection will not be great, perhaps will even be less than before, but the patient will have the benefit of not only an apparent but an actual result which may in some cases prolong life for many years.

At the present time, our conclusions amount to this, that in cases in which total extirpation cannot be performed we should under special circumstances advise gastroenterostomy; for example, when there are very marked disturbances (pain, vomiting), a fair degree of strength, and when the use of internal remedies has been futile. Occasionally, at the urgent desire of the patient and his relatives, to whom, for personal reasons, even a brief prolongation of life is a boon, we are constrained to consent. When the question of total extirpation arises, and the surgeon considers the patient's strength sufficient, this operation should be performed. Often enough, in such cases, we will be convinced of the futility of total extirpation and content ourselves with gastroenterostomy, but now and then the constellation of conditions appears so favorable that radical procedures are undertaken.

We now turn to *carcinoma of the intestines*, and in the case of carcinoma of the small intestine the laws and indications are the same as those which have just been described. The higher the situation of the carcinoma the more difficult is its total removal, as the duodenum normally is so fixed that the total extirpation of a tumor is not only extremely difficult and attended therefore with great loss of time but is combined with great danger for the patient. Lower down, as is proven by a few successful sarcoma operations, complete success has been attained. But the decision as to the most suitable time for operation meets with many obstacles.

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[1] *Boas*, "Diagnostik und Therapie der Magenkrankheiten," II, 4, Auflage, p. 222.

The conditions are more favorable in carcinomata of the large intestine and rectum. Whenever possible the radical method is here to be preferred to the palliative. Generally speaking, the conditions for total extirpation in the previously discussed carcinoma varieties are decidedly better than in carcinoma of the stomach; for, as we have stated, metastases usually occur late, the tumor being for a long time movable, and less technical difficulty is encountered at the operation.

In spite of this the results fall far below our expectations. According to Wölffler-Schloffer, up to the year 1896 the mortality in resections of carcinoma of the large intestine still amounted to 50 per cent. Yet the prognosis in regard to prolongation of life appears to be decidedly better than in high-seated carcinomata. Cases are on record of cure lasting seventeen years (Martini-Gussenbauer), of ten years (Czerny, a case of sarcoma), of eight and nine years (Körte), etc. I have a patient who had carcinoma of the cecum (adenocarcinoma) and whose recovery has lasted over five years.

Nevertheless, up to the present time such recoveries are rare. Most cases, even after a radical operation, perish in the next two years.

In case a radical operation cannot be performed two palliative methods of treatment remain: Entero-anastomosis and anus prœter naturam (colotomy). Unquestionably the first method is preferable to the other for obvious reasons. It also gives relatively good results—according to Wölffler-Schloffer the mortality is 20 per cent.—and prolongs life from a year to a year and a half. But here we must bear in mind not only that the patients live, but how they live. Although there are very favorable functional results, I have repeatedly observed, soon after the entero-anastomosis, renewed attacks of severe colic, marked loss of weight which had at first been increased, edema, and ascites.

In conclusion, *colostomy* will only come into question when, on account of the general condition, an immediate operation is necessary, therefore either when the strength is markedly reduced in the early stages of obstruction, or where debility has more fully developed. These conditions will usually be found associated. The indications for colostomy are closely related to the situation of the carcinoma. For example, in carcinoma of the cecum the production of a fistula of the ileum is a questionable proceeding, both from a cosmetic and a nutritive standpoint. The true domain of colostomy is found in carcinoma of the sigmoid flexure and of the rectum, for here the feces have acquired a consistence which makes it possible readily to remove them.

We now turn to the operative treatment of *carcinoma of the rectum* According to the seat, the development, the movability of the tumor, and the presence of metastases, the following procedures must be considered

Extirpation of the tumor, curettage, and the production of an artificial anus.

The ideal method is, naturally, the radical removal of the tumor (amputatio recti or resectio recti), but, only when the tumor is sufficiently movable, and when there are no metastases of distant organs, especially of the liver, is this possible. Enlargement of regional lymph-glands, however, forms no contra-indication to total extirpation. Since the performance of resection of the sacrum by Kraske (1882) high-seated carcinomata of the rectum have also been radically removed.

The results of these operations at the hands of different surgeons are far asunder, but in the last ten years they have become decidedly more favorable. According to Czerny's comprehensive statistics of the Heidelberg Clinic, which have the advantage of originating with him and which cover a long period of time, 152 rectal carcinomata were observed in the years from 1878 to 1891 and 109 radical operations were performed. Among 83 cases which were operated upon by the perineal method 3 died, = 3.6 per cent.; of 66 according to the sacral method, 9 perished, giving 13.64 per cent. The total mortality of 10 deaths in 109 cases is 9.1 per cent. Of 99 in whom radical operation was performed followed by a cure, 21 lived two years and more after the operation, 15, three years and longer, 13, four years and longer, 8, five years and longer; among these one case lived for 18 years, one case for 16 years, four cases respectively thirteen years and nine months, eleven years and six months, eight years and nine months, and six years and nine months. The frequency of relapse after extirpation is calculated differently by different authors. The figures vary between 41.6 and 73.3 per cent. Czerny maintains that 20 to 25 per cent. of those in whom radical operation is performed continue free from relapses for two years, and the majority of them are permanently cured. The sacral method in particular diminishes the danger of relapse, as the lymph-glands situated in the excavatio sacralis are likewise removed.

The functional results vary according to whether the sphincter is retained or removed. In the former case, the functional results, *quoad continentiam recti,* are very satisfactory. In the latter, the condition of the patient is most unpleasant, as he can retain neither gas nor fluid feces, and it is therefore necessary by diet and drugs to make the feces compact.

Where a radical operation is impossible, as is unfortunately often the case, there are two methods, curetting and the production of an artificial anus—the one direct, the other indirect—by which we diminish or arrest the symptoms of stenosis. The curetting which may be performed with instruments, or, still better, with the hands, is best adapted to deep-seated carcinomata with special implication of the posterior wall of the rectum. When the tumor is high-seated it is best not to use this method as, under some circumstances, more harm than good is done. The same is true o deep-seated carcinomata in the anterior wall of the rectum.

The most suitable and, relatively, the least dangerous measure for th removal of the symptoms of stenosis is unquestionably colostomy. Thi method most certainly relieves the irritation of the tumor, and cases ar on record of prolongation of life for two or three years after the production of an artificial anus.

In regard to the indications for colostomy the opinions of physician and surgeons are wide asunder. While colostomy is frequently employe in England and France, in Germany, like all fistulous operations, it appear to have lost favor in the last few years. We may here speak of a relativ and an absolute indication for its performance. The indication is relativ if, while a fair intestinal passage remains, the patient steadily loses i weight and strength, is tormented by pain, and passes much pus and bloo In such cases we are decidedly in favor of operation, provided intern remedies have proven ineffectual. As long as evacuations at all sufficie are brought about by mild dietetic measures or by purgatives, we do n advise colostomy. With a suitable diet I have kept well advanced cas of carcinoma of the rectum alive just as long as by the production of artificial anus.

Colostomy is absolutely indicated when complete intestinal occlusi occurs or is threatened. In this case it is best not to wait so long th the patient's strength is diminished by vomiting, pain, and high-grad meteorism. The functional results of colostomy are satisfactory, provid we are careful to produce compact feces and the fistulous opening is k well closed with a hernia bandage. We must consider, too, that ma patients naturally find the daily manipulation of their intestines and the dejecta exceedingly repugnant. It is, therefore, the duty of the ph cian and the surgeon, in order to prevent subsequent regret, to expl to the patient in no uncertain manner before the operation the light shade aspects of the anus præternaturalis.

DISPLACEMENTS OF THE ABDOMINAL VISCERA AND OF THE HEART

By F. HIRSCHFELD, Berlin.

INTRODUCTION

Until about twenty years ago a displacement of the viscera was generally regarded as a rarity. It is true that at the beginning of the nineteenth century celebrated clinicians such as Esquirol had devoted much consideration to displacements of the abdominal viscera, and had even recognized the connection between displacement of the colon and the development of mental disease; but, toward the middle of the century, these views were no longer considered tenable, as appears from the works of Griesinger, Wunderlich, Canstatt and others. Displacement of the colon was looked upon as unimportant, that of the stomach could not be detected by the ordinary clinical methods of the day, and only that of the kidneys was considered, this, however, being regarded as a rare occurrence since it was only occasionally noted by anatomists. The reason for this may be found in the technic of autopsies, as well as in the fact that in most cadavers the organs have regained their normal position by the bodily rest which usually precedes death. It is true that in isolated reports abnormal movability was occasionally pointed out, but general attention was directed to this only about in 1880, in Germany, by the labors of Landau, Leube, Ewald, L. Kuttner, Litten and Meinert, and in France by Glénard, Féréol and Cuilleret. In 1890, Virchow demonstrated that a change in the position of the abdominal viscera, and particularly of the intestines, could be detected in the majority of persons. Glénard did much to promote the recognition of these changes in position as a pathologic condition by creating the name, *enteroptosis*. His investigations, however, soon led him to a path on which he could no longer be followed. His original view that enteroptosis is a sharply characterized substantive disease (entité morbide) was not at once generally accepted, but in the main was considered justifiable. It was admitted that in many persons a moderate degree of downward displacement of the stomach, a unilateral movability of the kidney, and a displacement of the colon could be demonstrated, and the embodiment of these phenomena in one clinical picture appear all the more warranted since they presented themselves in nervous, we females, usually anemic, therefore in persons who in their external appe ance already presented the same type. Glénard believed that downwa displacement of the colon, particularly of the right portion of the tra verse colon, was chiefly responsible for the production of these disturbanc On the other hand, in the development of displacement of these orga he was but little inclined to take into consideration mechanical conditio flaccidity of the abdominal walls, the disappearance of fat in the abdo inal spaces, the influence of external pressure from lacing, etc. The i portance of these factors in the etiology of enteroptosis was especia demonstrated by Landau, Meinert, and Dennig, and was proven by t fact that in many persons, and under certain conditions, a loosening the attachments and the descent of one or more organs was possible. T anatomical prior condition may be favorable to a displacement in o case, still more so in another, but it is difficult to regard this weakn of the ligaments as the foundation of a disease, particularly as it occ in most persons without producing symptoms. Thus we see floating kidr appear in one person by the development of asthma and emphysen because of the disappearance of fat in the abdominal cavity, and, perha because the flaccidity of the abdominal walls which increases with diminishes pressure in the abdomen, and thus the firm position of kidneys is so far weakened that under the influence of shock to abdominal cavity (as in prolonged paroxysms of coughing) they fin descend; in other cases, however, we note particularly how the hear limited in its movability by the formation of empyema in the lungs, subsequently the phenomenon of "displaced heart" disappears. Only minute consideration of all the mechanical conditions will enable u understand clearly the genesis of these changes in position and t influence upon the functions of the various organs.

By some clinicians, such as Stiller and Obrastzow, enteroptosi splanchnoptosis is to a certain extent regarded as the sign of degenera The imperfect cartilaginous attachment of the tenth rib to the th (costa decima fluctuans), frequently found in such persons, is looked as proof. A certain justification must be admitted for this view, s undoubtedly, a displacement of the abdominal viscera is found in a gr percentage of persons predisposed to nervous affections than in the mally strong. On the other hand, we must bear in mind that in ne persons a lessened muscular activity, a weaker muscular structure, probably a more flaccid ligamentous apparatus, are generally to be exp Therefore, that a displacement of the internal organs may readily does not appear remarkable, and this circumstance naturally explai greater distribution of enteroptosis among the nervous. Whether, in tion, a certain, and perhaps also a hereditary, predisposition can sumed is extremely difficult to decide. Very likely a feeble ligamentous apparatus in the internal organs and certain peculiarities in the structure of the same may be hereditary, as well as a special predisposition of the bony skeleton; but, for the development of enteroptosis, perhaps, just as in the development of kyphoscoliosis of the vertebral column, a number of external influences are necessary. Therefore, we do not agree with Glénard who, in accepting the French views regarding l'arthritisme, propounds a theory according to which most diseases are related to enteroptosis, and a disturbance in the activity of the liver which is not described (l'hépatisme) is the most significant symptom.[1]

Although certain uniform factors are decisive in the development and treatment of the various displacements of the organs, it appears to be most necessary for the correct understanding of the subject under discussion to describe the displacement of each individual organ separately.

Most important are the displacements of the stomach and the kidneys. Displacements of the spleen and of the liver are generally rare, those of the colon and heart more frequent but of less clinical importance.

DISPLACEMENT OF THE STOMACH (GASTROPTOSIS)

Reports vary as to the frequency of gastroptosis. According to Meinert,[2] in Dresden the majority of women have a displacement of the stomach while, by estimation, only about 5 per cent. of men show an alteration in the position of this organ. Similar proportions have been reported by Dennig[3] in the population of Württemberg. From his figures it is very evident in which years of life gastroptosis is specially prone to appear.

Among 2,000 persons who presented themselves at a Stuttgart Polyclinic on account of various ailments not connected with disease of the stomach,

in 29.7 per cent. of the men,
in 75.4 per cent. of the women,

a displacement of the stomach was determined.

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[1] The views of Glénard, except those in his earlier publications (*Lyon médical*, 1885 et 1887), are found in his comprehensive work: "Les ptoses viscérales. Diagnostic et nosographie." Paris, 1899, 962 pages. Compare also Le progrès médical, 1899, I, page 320; 1900, I, page 225; 1902, I, No. 2.

[2] *Meinert, Sammlung klin. Vorträge.* N. F., Nr. 115 u. 116, Leipzig, 1895; *Centralblatt f. innere Med.*, 1895, Nr. 43 und ebenda, 1896, Nr. 13 u. 14.

[3] *A. Dennig, Württembergisches med. Correspondenzbl.*, 1903, Nr. 18.

and the embodiment of [illegible] gastroptosis was present [illegible] all the more warrant[illegible] females, usually [illegible] ance already present[illegible] displacement of [illegible] verse colon, was [illegible] On the other hand, [illegible] he was but little [illegible] flaccidity of [illegible] inal space, [illegible] portance of [illegible] demonstrated [illegible] fact that in [illegible] the attach[illegible] in girls [illegible] anatomic[illegible] case, still [illegible] of the [illegible]

	In Men.	[illegible]
	Per cent.	[illegible]
[illegible]	[illegible]	
[illegible]	[illegible]	
[illegible]	[illegible]	
[illegible]	[illegible]	
[illegible]	[illegible]	
[illegible]	[illegible]	
[illegible]	[illegible]	
[illegible]	[illegible]	
[illegible]	[illegible]	
[illegible]	[illegible]	[illegible]

[illegible] years, in 20 per cent.,
[illegible] " " 57 "

In most [illegible] seen that gastroptosis is mu[illegible] [illegible] This displacement probably [illegible] as a consequence of wearing a co[illegible] [illegible] in the following decades flacci[illegible] [illegible] until, finally, almost all [illegible] [illegible] Dennig the same deleteri[illegible] [illegible] to a habit very common [illegible] [illegible] of tying the skirts [illegible] [illegible] When the hips h[illegible] [illegible] in woman, tightness ab[illegible] [illegible] skirt from slipping down. [illegible] as is the rule with ch[illegible] [illegible] of age.

[illegible] by the results of [illegible] [illegible] of wearing a belt to [illegible] [illegible] is distinctly [illegible] [illegible] were narrow[illegible] [illegible] a normal position [illegible] [illegible] suspenders.

[illegible] position. M[illegible] [illegible] with a vertically [illegible] [illegible] situated, but that [illegible] [illegible] of the st[illegible] [illegible]

[illegible] gastroptosis in Saxony, [illegible] [illegible] rise to much specul[illegible] [illegible] and believe that the [illegible] amounts of gases developed from the carbonic acid employed for distending the stomach abnormally displace this organ, and probably also give no well-defined idea of the size of the stomach so inflated. According to Meinert, in the above investigations

8 grams of sodium bicarbonate,
6 " of tartaric acid,

were employed; this was dissolved in a little water and taken at once or in divided doses rapidly succeeding each other. Gastroptosis was assumed whenever the pylorus could not be recognized at the scrobiculus cordis, or was situated more deeply; the greater and lesser curvatures of the stomach must have descended at the same time.

As large doses of this mixture will produce nearly 3 liters of carbonic acid, the criticism is justifiable that the normal stomach which, as a rule, has merely a capacity of about 1½ liters, is immoderately distended and perhaps also is drawn somewhat downward. Dennig employed a large double bellows with which he inflated the stomach after introducing the stomach-tube. With this method, as I have fully convinced myself, particularly with tense belly walls, it is necessary to exert considerable pressure if the boundaries of the stomach are to be sharply defined. We must, however, remember that neither in powerful men nor in children does this downward displacement of the distended stomach ever take place. We are thus forced to conclude *that this property of the stomach or, rather, of the pylorus, to descend upon immoderate inflation,* is a pathologic deviation, and to a certain extent indicates a predisposition to gastroptosis.

In the description of the development of gastroptosis from the influence of gastric catarrh, general weakness of the stomach, etc., I shall attempt to show that, to a certain extent, the same process takes place as in the downward displacement of the organ artificially inflated by carbonic acid gas.

The purely mechanical course may best be understood by some illustrations.[1]

In Fig. 11 we see a stomach in its normal position. The pylorus is situated at a point almost directly behind the right arch of the ribs, where this is crossed by the tip of the ensiform process upon a horizontal plane (Meinert). The axis of the stomach more nearly approaches the horizontal than the vertical. The pyloric portion of the stomach is in the main situated in the scrobiculus cordis.

In Fig. 12 we see the so-called vertical position of the large normal stomach when inflated. The pylorus can no longer be detected in the pi of the stomach. The greater curvature is decidedly lower than the umbili cus. The axis of the stomach is in a vertical rather than a horizonta position.

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[1] These illustrations have been taken from the "Diagnostic Lexicon" of *A. Bum* and *Schnirer*, Vienna, 1884, III, p. 111. Presentation of gastric examination by Rosenheim. My description of the individual figures differs somewhat from that given by Rosenheim.

Fig. 13 shows a somewhat later stage. The greater curvature has de scended lower. The cardia may be recognized in about the same area a

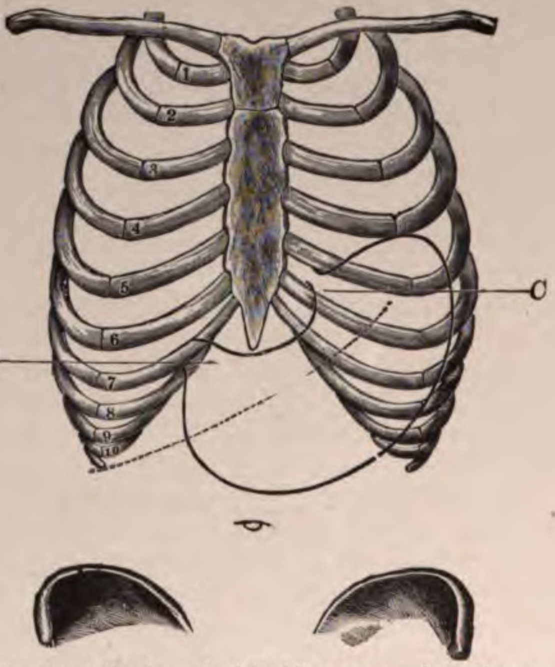

FIG. 11.—Normal situation of the stomach; dotted line, ..., shows the margin of the li

in the normal stomach. Especially noteworthy is the great bulging of pyloric portion of the stomach downward and to the right. As the musc of the stomach which chiefly propel the chyle are situated around pylorus, an enlargement of the stomach to the right, which may be tinctly demonstrated by percussion, clearly denotes a weakness of muscular apparatus and beginning insufficiency of the same,[1] altho gastric dulness appears, in the main, to be but slightly increased.

In Fig. 14 we see a decidedly enlarged stomach. Such a condi may have been caused by gastroptosis and subsequent insufficiency as as by a mere descent from gastrectasis. A decision cannot be based a upon these pictures; for this purpose the consideration of all the po necessary in an examination is called for.

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[1] *Rosenheim*, "Krankheiten der Speiseröhre und des Magens," I. Aufl. 1891; *Deutsch. med. Wochenschr.* 1896; und *W. Michaelis*, *Zeitschr. f. klin.* XXXIV.

A somewhat different condition is shown by Fig. 15, which is taken from Bollinger's book.[1]

This illustration of Bollinger, first of all, shows distinctly how the vertical position of the stomach with constriction of its pyloric portion occurs from the external pressure of tight lacing, just as this acts upon the liver. A vertical position of the stomach without dilatation is frequently not considered pathologic by the anatomist, and therefore we often see illustrations of a vertical stomach in books upon normal topographic anatomy.

How do the effects of a downward displacement of the pylorus, of gastroptosis, become noticeable during life?

In a normal person this occurs by downward pressure on the liver and pylorus in consequence of tight lacing around the waist, and, except for slight discomfort which, perhaps, is only due to pressure upon the skin, the activity of these organs is usually but slightly affected. It is true the passage of the chyle from the stomach into the duodenum is probably impeded, but we know that the smooth muscles of the intestine rapidly hypertrophy after the addition of obstructions in the intestinal canal, and thus by increased labor bring about compensation. The majority of healthy

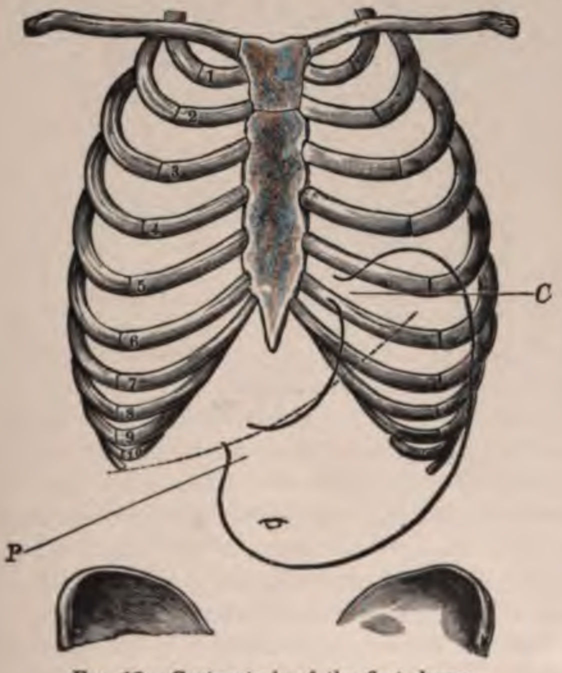

FIG. 12.—Gastroptosis of the first degree.

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[1] *O. Bollinger*, "Atlas und Grundriss der pathologischen Anatomie," I., page 193, 2. Aufl. München, 1901.

young girls, therefore, notice no immediate, injurious effect from th wearing of a constricting corset which simultaneously presses down th liver and the pylorus. It is different, however, if the stomach of th

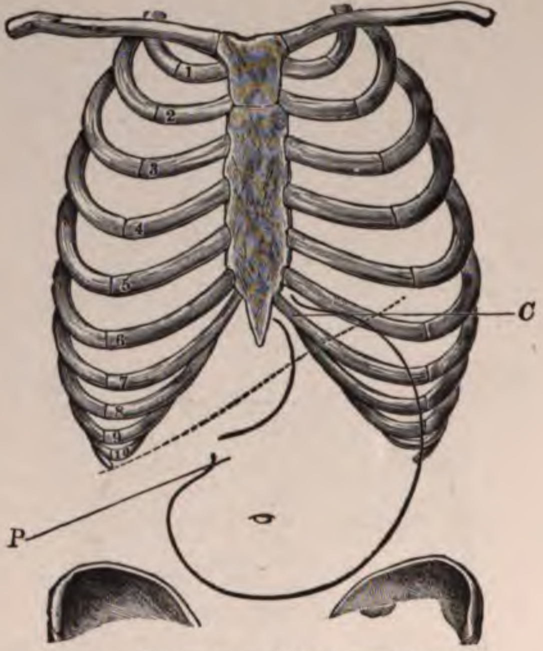

FIG. 13.—Gastroptosis of the second degree.

person in question has previously shown a tendency to disturbance, o a general weakness of the system exists.

These disturbances most frequently occur in young, anemic, po nourished, growing girls. Under these circumstances, the stomach can so readily compensate the obstruction to the propulsion of chyle by unfavorable position. Decided insufficiency readily takes place. The sy toms of the patient all distinctly point to the fact that the corset is disturber of the peace, for, after discarding it, decided amelioratio noted. Gradually the gastric catarrh disappears, and, on resuming corset, the patient readily becomes accustomed to her instrument of tort

In these cases, therefore, a slight gastroptosis persists which us cannot be determined by the ordinary clinical methods of examina unless it be by decided inflation of the stomach. As a rule, symp appear only when an error in diet produces an acute gastric cat While the consequences of such a gastric disturbance in patients wi normal stomach soon disappear under suitable treatment, in those a latent gastroptosis a high-graded insufficiency of the stomach occa ally develops. A loud, splashing sound most distinctly reveals the enl ment of the stomach. The greater the distention of the stomach in the course of the affection, the more difficult, naturally, is the propulsion of chyle into the duodenum. The affection, therefore, spontaneously forms a vicious circle. An immoderate dilatation of the stomach is also promoted when the belly wall is very flaccid. Even in young girls who are unaccustomed to bodily labor, the muscles exerted in the abdominal press form only a flaccid, thin wall. The activity of the abdominal muscles is still further impaired in women by successive labors, the consequent pendulous abdomen described by Landau[1] becoming particularly noticeable.

The persistence of gastric catarrh or gastric insufficiency in gastroptosis, however, leads to other dangers. Being insufficiently nourished the organism is forced by metabolism to the combustion of its body substance, the fat in the abdomen and the mesentery being first utilized for this purpose. Here we note the well known fact that in an antifat treatment

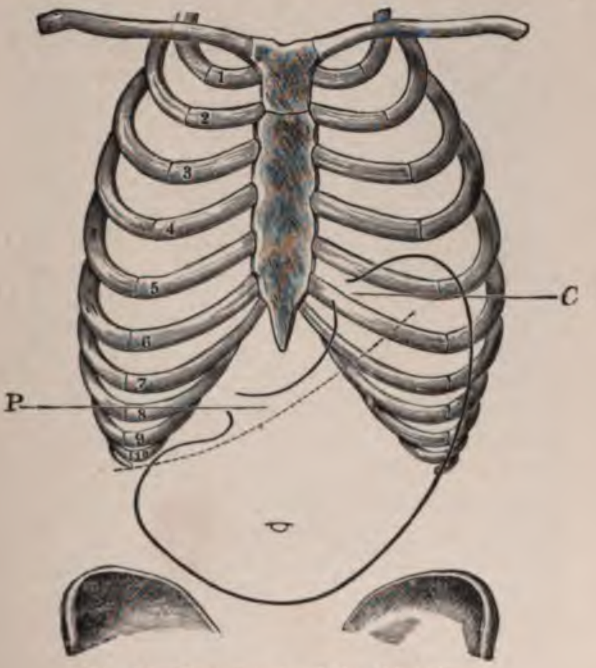

FIG. 14.—Gastroptosis of the third degree.

a decrease in the circumference of the waist is most perceptible. The loss of fat accumulated in the abdominal cavity makes itself felt in the same way as a weakening of the abdominal walls. The position and attachment of the organs are no longer secured by simultaneous pressure, but these depend upon the suspensory ligaments which may readily yield. In severe diseases, such as gall-stone colic, gastric ulcer, or gastr this dilatation of the prolapsed stomach becomes noticeable m frequently than after simple gastric catarrh. Its manner of de is interesting, and is as follows: I have repeatedly heard youn who have recovered from an appendicitis operation state that ir

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[1] *Landau, Verhandl. der Berl. med. Gesellschaft*, 1890.

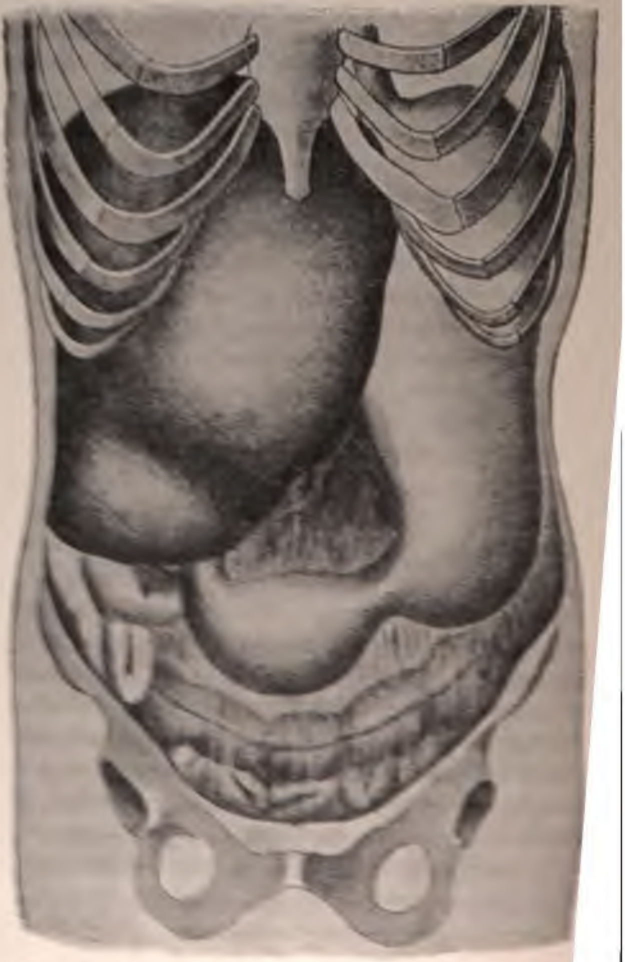

Fig. 33.—Constriction of the right lobe of the liver and of the py

few days after they had left their beds they observed an i circumference of the abdomen, which was confirmed by the amination of the physician. In these cases there was no abd but an enlargement and displacement of the stomach was any symptom of gastric disease. This was explained by t appearance of fatty tissue in the abdominal cavity, since question had lost from 11 to 16½ lbs. in weight. A few days after getting out of bed, and being in an upright position, they were well enough to take large quantities of food, and this had caused the over-burdened and distended stomach with all of the other viscera to descend.

That occasionally a decided displacement of the stomach may be determined in kyphoscoliosis is readily comprehended. In these cases, also, the prolapse usually occurs without producing any disturbance in gastric activity (Fleiner[1]).

Lordosis of the vertebral column, which sometimes develops to a decided degree in women predisposed to it if they wear high heels, produces a condition in which the pressure of the viscera dilates the abdominal walls downward and anteriorly, and the internal organs themselves drop (Cséry, Korányi, and Meltzing[2]).

In some diseases, for instance, tuberculosis, the effect of mechanical shock makes itself felt in the abdominal cavity. The paroxysmal shock to the thorax and abdominal wall produced by cough must assist this process, particularly when the factors we have mentioned which favor the descent of the stomach have already been active.

From these descriptions it is obvious that gastroptosis may be looked upon as a disease in only a small number of cases when, from any complication, the compensation is lacking which would otherwise be produced by increased activity of the muscular structure of the pylorus. In regard to the frequency of these disturbances, no absolute general rules can be laid down.

Gastroptosis and enteroptosis are most frequently met with in patients suffering from gastric disease. Thus Einhorn[3] reports that within a few months he found 9 cases among 141 male patients with gastric disease, and among 92 women he found these changes 32 times. It is true that the more vigilantly we search for changes in position, the more frequently we will diagnosticate them in a greater or less degree. Often the appearance of hysteria or neurasthenia leads us to suspect gastroptosis. It may then be doubtful which affection existed first.

SYMPTOMS

Symptoms of gastroptosis may be entirely absent. In young girls who press the pylorus down by wearing a corset, possibly because of the increased difficulty with which the chyle is propelled into the duodenum, eructations will often occur, and the peculiar rumbling or gurgling frequently heard in women who lace very tight is declared by Fleiner to be

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[1] ***Fleiner*, *Münch. med. Wochenschr.*** 1895, Nr. 42–44.

[2] ***Korányi*, *Berl. kl. Wochenschr.*,** 1890, Nr. 31; *Meltzing, Volkmann'sche Vortr.* 1896; *Csèry, Wiener med. Wochenschr.*, 1901, Nr. 28.

[3] ***M. Einhorn*,** "Die Krankheiten des Magens," Berlin, 1898, page 270.

ɹɪ stenosis due to compression of the stomach. In most ca
however, as has been stated, symptoms are either absent, or these r
signs are regarded by healthy persons as due to a disturbance of gas
movement. In weak, anemic persons with a tendency to chlorosis
constriction of the stomach may exert such a deleterious effect that
only the signs of gastric catarrh but also manifold nervous disturba
may present themselves. The symptoms due to gastroptosis are then cl
intermingled with those of chlorosis, of general debility, and of nerv
ness, but are, in the main, attributed to chlorosis; they may continue
weeks, months, or even for years. Quite properly Meinert attributes
old axiom, founded upon experience, that marriage is the remedy
chlorosis to the fact that when pregnancy occurs the growing uterus f
the distended stomach upward again.

The symptoms which subsequently arise, provided no intercurrent
ease suddenly produce marked insufficiency of gastric activity, as in
manner I have described, resemble those which generally appear in
sons suffering from gastric catarrh. A conspicuous symptom in
patients is the great weakness and debility which cannot be acco
for by any preceding error in diet. In the horizontal position, espe
in the right lateral, the symptoms ameliorate, for then the propulsi
food goes on under the most favorable mechanical conditions. Vom
is rarely mentioned in the history, but is most frequent at the onset
very rare in the later course. Incessant vomiting throughout the
period of the disease might therefore be utilized as excluding gastrop

The patients almost invariably complain of marked constipation
which various causes are assigned. In the first place, a displaceme
the colon, particularly of the mesocolon in which the thin, fluid
first become more compact, may cause kinking and, naturally, a retard
of the feces. However, too much stress is not to be laid upon thi
cumstance, since, after the removal of other causes, and in spite o
persistently abnormal course of the colon, regular fecal discharges a
always take place. It is significant that these women—and with
we usually deal in gastroptosis—generally prefer easily digested food
leaves but little residue in the intestine. We know from physiology
with food consisting chiefly of meat and wheat bread the fecal m
twenty-four hours amounts to only 100 to 130 grams of moist, and
20 to 30 grams of dry, substance, while with a diet rich in cellulose,
fore with the ingestion of much rye bread, the feces amount to 400 t
grams of moist, and 60 to 80 grams of dry, substance. As the int
food in gastric affections is generally even less, and accordingly there
residue, a fecal evacuation every two or three days appears quite n
Moreover, a pendulous abdomen decidedly decreases the force of t
dominal press, so that small fecal masses formed from easily digeste
are only evacuated by great effort. Finally, in my experience, th

of muscular exercise on the part of most women adds much to torpidity of the bowels, which therefore cannot be looked upon as entirely the consequence of gastroptosis, coloptosis or enteroptosis.

Among the objective symptoms, a loud splashing sound in the umbilical region is noted by the physician. This sign is so characteristic that when absent in repeated examinations one or two hours after the ingestion of fluid, insufficiency of the displaced stomach may be denied. The importance of the succussion sound in judging of the gastric activity has lately been the subject of much controversy. Although I am willing to admit that this symptom may occasionally be observed in thin, healthy persons, nevertheless I coincide with Stiller and Kuttner[1] in the view that, under these circumstances, the gastric wall is incapable of contracting strongly enough about its contents. In gastroptosis the splashing sound is usually detected around the umbilicus, often, however, below this region.

Among other signs epigastric pulsation, which is emphasized by Glénard, must be considered. It is not of the same diagnostic import as the succussion sound, for, in the first place, when the abdominal walls are thin and the pylorus is situated in the scrobiculus cordis, the pulsation of the vessels may sometimes be felt through the gastric walls, and secondly, when the abdominal walls are rich in adipose tissue, pulsation is unrecognizable notwithstanding the existence of gastroptosis.

Another symptom, a feeling as of the pressure of a girdle in the gastric region, the "corde colique," leaves us completely in doubt. Glénard believes this band to be the colon, while, according to Ewald, the area of resistance, which, however, can rarely be felt, is to be looked upon as the pancreas. This fact is not generally known; it is, however, occasionally of great practical value, for, otherwise, upon palpation in this region, and when there is a disturbance of gastric activity, we might mistake this resistance for a neoplasm. I know of a case in which such an error led to a laparotomy being performed—a state of things naturally not very desirable, yet not so serious as the inverse assumption that a neoplasm is only the head of the pancreas.

The most signal proof of gastroptosis, or that the disturbances present are chiefly due to displacement of the stomach, is furnished us by the movability of other abdominal organs, particularly of the kidneys. In how far it is possible to palpate the kidneys will be discussed later. In almost every marked case of gastroptosis it is possible distinctly to palpate the kidneys, at least the right one, upon the posterior wall of the abdomen. Often one or both kidneys are movable in the abdominal cavity, or they are shown to be fixed in certain areas.

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[1] *Berliner klin. Wochenschr.*, 1901, Nr. 50.

DIAGNOSIS

Naturally the diagnosis is assisted when the walls are very flaccid. In the majority of cases of gastroptosis the distention of the stomach is so marked that the findings appear to be entirely out of proportion to the symptoms, which have usually existed but a short time. In gastrectasis which develops in consequence of some other gastric affection, derangement of the activity of the stomach has gradually become marked. Finally, we must remember that a displacement may have occurred secondarily from the cicatrix of an ulcer or a neoplasm which had narrowed the stomach, and that dilatation had subsequently occurred. In practice, therefore, the proof of a displacement is naturally much less significant than the recognition of some other obstruction to the propulsion of the ingesta through the pylorus. For this reason, in the examination of a patient suffering from gastric disease, I must advise against a great inflation of the stomach, provided we desire only to determine gastroptosis or the tendency to displacement. *In my experience, only in hysterical, very nervous persons, or in those debilitated from disease of some other organ, will gastroptosis cause a decided disturbance in nutrition.* On the contrary, examination should reveal the cause of the compensatory disturbance in the activity of the displaced stomach. According to the age, the sex, and the general appearance of the patient, we must search for *gastric ulcer*, for *malignant disease of the gastrointestinal canal*, for *cholelithiasis*, or for *pulmonary tuberculosis*.

TREATMENT

In the treatment, prophylaxis plays the first rôle. By prohibiting lacing the chief cause of the development of gastroptosis, as well as displacement of other organs, will be removed. Probably every woman will assure the physician that she does not lace too tight, although an examination of the skin will reveal distinct lines about the waist due to the constricting corset. In regard to these views there is little unanimity between physicians and their women patients. A corset of stiff material worn by those anemic women, weak in muscle, who form the majority of the well-to-do classes, may have the advantage that it gives a certain support to the weak vertebral column which is lacking in strength of muscle and will remedy defects in the carriage of the body. But, as a rule, the physician must insist that the corset be not worn too tight, nor the skirts suspended from the waist. This danger is greatest in girls from fourteen to sixteen years of age whose hips have not yet sufficiently developed. The skirts must be suspended from the shoulders like the trousers of men, and a belt must not be worn. It is well known that the "dress reform," which has already become somewhat general in Germany, is directed toward this purpose. Unfortunately, stout women refuse to abandon the corset, because without it the waist appears to them to be too large, and the bust is not sufficiently supported. It is to be hoped, although it is by no means certain, that in this respect common sense will finally prevail over vanity and the present standards of beauty.

On account of the tendency of the vertebral column to marked lordosis, the wearing of high-heeled shoes should be prohibited, the height of the heel being limited to 2 or 3 cm.

The formation of a pendulous abdomen in consequence of childbirth may be prevented by maintaining the recumbent posture as long as possible after labor, and on rising from bed the flaccid abdominal walls should be firmly supported by a well-fitting bandage.

The most effectual means to prevent the displacement of organs probably lies in the education of the female from infancy as to the value of physical exercise. With this we might hope that the abdominal, as well as all the other muscles of the body, would attain their fullest development, and that the wearing of sensible clothing by the majority of women would prevent many other disabilities of civilized people.

If a displacement of the stomach has already occurred, yet no symptoms are present, treatment may appear to be superfluous. Nevertheless, in such cases I must advise prophylactic interference and the interdiction of tight lacing, especially if occasional symptoms, such as loud gurgling in the intestines, have already been noted. If there are distinct signs of insufficiency of the stomach, a binder which supports the lower parts of the abdomen is serviceable. An abdominal bandage lately advised by Ostertag[1] is especially valuable for this purpose. In women the symptoms sometimes disappear immediately on its use.

In advising an abdominal binder it must be borne in mind that it would be the height of folly to forbid, on the one hand, the constricting corset, and then, for the opposite effect, to recommend an abdominal bandage which compresses the organs from the waist downward.

According to the statements of Landau and Bardenheuer, there are stays supplied by instrument makers which exert no special pressure about the waist, and to this the abdominal bandage for the lower parts of the abdomen is already attached. I advise these corsets in antifat cures even if there is no gastroptosis, since in hyponutrition, as already remarked, fat disappears from the abdominal cavity with comparative rapidity and, therefore, the condition for displacement of organs is already present. Bial[2] has lately proven by the illumination of the organ that a prolapsed stomach cannot be supported by a bandage. He also relates his experiences, according to which the desired result may be attained by suitable hydrotherapeutic and dietetic measures. Nevertheless I advise the employment of these abdominal supporters. In the first place, in women the weakened abdominal wall is strengthened, and thereby the danger of a marked displacement of the abdominal organs is averted. Moreover, it is quite clear to me that not only does the motor activity of the stomach improve, but the action of the bowels is decidedly facilitated. The descent of the diaphragm, which by the abdominal press forces the abdominal viscera downward, encounters a stronger resistance in the abdominal bandage than in the flaccid belly walls, which, when strong pressure is exerted, invariably become more distended.

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[1] *Ostertag, Monatsschr. f. Geburtsh.* u. Gyn., XV.

[2] *Bial, Verh. d. Congr. f. innere Med.*, 1897, p. 521.

Dietetic rules form an important part of the treatment. From a purely mechanical standpoint the difficulty of propelling the ingesta from the stomach is at once obvious. v. Mering[1] has lately pointed out that in the right lateral position the stomach is most rapidly emptied, and, under the conditions here discussed, we should utilize this fact. In debilitated persons, a rest of one or two hours in the right lateral position after the principal meal should certainly be advised.

Furthermore, it is unwise to permit too copious meals. At least while active symptoms are present, several small meals must take the place of the chief meal. Oertel and Schweninger's advice to the obese not to drink while eating, which they erroneously regarded as an important law, is, on the contrary, well adapted to gastric patients, and particularly to those suffering from gastroptosis.

Under some circumstances we may attempt to hasten the evacuation of the stomach by massage. In most cases I believe this effect is to be attributed to autosuggestion. Nevertheless, I have several times seen such favorable results that I frequently advise it, and the more so since it may be readily practised by the patients upon themselves. The hand rests upon the left arch of the ribs, and about this point rotary motions are made from the left below to the right upward. Massage by another person I do not advise, because, particularly with movable kidneys, there is danger that these may be irritated—a danger which cannot be excluded.

I am, therefore, decidedly opposed to the advice given by some authors to treat gastroptosis by a course of massage of from six to eight weeks, in which the massage is given one or two hours before eating. This will not bring about a true cure of gastroptosis, and a temporary insufficiency of the musculature of the stomach may more certainly be made to disappear by other means.

In the choice of food the same rules are operative which are applicable to all patients suffering from gastric disease. When there is marked disturbance of gastric digestion small quantities of milk may first be permitted, and are very serviceable as easily digested, rich, nutritive products. It is not advisable to give more than one liter of milk in the course of the day; more than this will over-burden the stomach. If there is repugnance to milk, cream may be given, more or less fatty according to the severity of the condition, and some tea. Zwieback, wheat bread, lean meat, eggs, green vegetables are the foods which next come into question. We must be careful with those foods which readily generate fermentation and gas in the stomach, therefore those especially rich in sugar. Naturally, the individual taste and the experience of special patients must here be taken into consideration. In regulating the bowels we must always consider whether a food shows a strong tendency to generate gas. In constipation we are generally inclined to advise foods rich in cellulose, consequently bread rich in gluten. The cellulose of gluten at first mechanically stimulates the intestine, subsequently it undergoes decomposition in the intestine by the activity of bacteria, and thus volatile acids and gases are formed. In gastroptosis, on account of the pendulous belly which usually coexists, gas production is probably already present and very disagreeable to the patient, so that, under these circumstances, the physician must be cautious. Here fruit, stewed fruit, and acid fruit wine, buttermilk or milk sugar lemonade are frequently preferable to the coarser varieties of rye bread.

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[1] *v. Mering, Therapie der Gegenwart,* 1902, Heft 5.

As in all displacements of organs we must consider in each individual case whether it is wise to attempt a "fattening cure" by profuse nourishment, so that the fat which has disappeared from the abdominal cavity may again accumulate there. I believe that this question should generally be answered in the affirmative. Patients with an affection of the stomach are usually so debilitated that even a gain in weight of a few pounds is worth our efforts, because this furnishes the best proof of the retardation of the pathologic phenomena. In all cases of hypernutrition prolonged rest is absolutely necessary, for we must never forget that the displaced stomach, even more than the normal stomach, is burdened by the ingested food. The more food, therefore, the patient eats, the less should the stomach labor under unfavorable mechanical conditions. As already stated, it is not enough that the patient assume the lateral position for some time after eating, but for the whole day, uninterruptedly, or at least for the greater part of the day, he should remain in bed or upon a lounge. The too free consumption of milk—over three liters daily, according to the advice of Weir Mitchell—as was formerly and is even now the general rule, can only aggravate the insufficiency of the displaced organ by overburdening the stomach. On the other hand, easily digested fats, such as cream, butter, bacon, or some carbohydrates, such as milk sugar and mannite, should be employed. Alcohol[1] can rarely be dispensed with in the form of brandy, good wine, or even as beer. But only slight quantities of the last named stimulant should be allowed, and never more than c.c. at a meal. Brandy may be advantageously given with milk, or with cream provided it be not too fat. Not only the property of alc as a nutrient, but also its stimulating effect upon the gastric activit especially desirable (G. Klemperer).

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[1] Compare *F. Hirschfeld*, "Die Ueberernährung und die Unterernährung," Frankfurt, 1897, p. 43 u. f.

In gastroptosis I have made but little use of drugs to improve the n tion. Sodium bicarbonate or magnesia, about as much as will cover tip of a knife, frequently lessens the gastric symptoms. Hydroch acid, as a rule, unquestionably has a deleterious effect. A favorable i ence is usually obtained by the employment of bitters; and I have quently used the following prescription:

℞ Tinct. Cinchona comp........................... 20.0
Tinct. Nucis Vomicæ............................ 5.0
30 drops in water or brandy three or four times daily.

In gastroptosis a mineral spring treatment is naturally not advis Even in Carlsbad, particularly in young, nervous persons, such treat of these cases is often ineffectual. A residence in a place of high alt is more beneficial, and care must be taken, especially at first, to too great bodily exertion, or to see, at least, that it corresponds wit strength of the patient. In nervous patients it is easily understooc rest in a sanatorium, or a residence with surroundings of beautiful sc will have a favorable effect. Sea baths, also, on account of their sti tion of the appetite, are worthy of consideration in some cases, b physician should always be consulted in these instances. In Ge bathing in the Baltic Sea is generally more beneficial to such p than in the North Sea.

Finally, in gastroptosis, as in so many other diseases, hydrot may be employed, and is particularly adapted to the many nervous toms which develop in connection with all gastric affections.

Buxbaum [1] and Winternitz advise quick, cool Sitz baths, while (Matthes, Boas [2]) have seen favorable results from a needle douch may be used either cold or, like the so-called Scotch douche, cool an alternately. In this treatment, other measures may occasionally the local douche, as a lukewarm full bath, or general friction entire body.

For the sake of completeness I shall only mention that electric ment of the stomach, the faradic as well as the galvanic current, sionally employed in gastroptosis. I saw favorable results in a which almost all other remedies had proven ineffectual. The patient was a lady, exceedingly nervous, who complained of spasmodic pain several hours after taking food.

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[1] *Buxbaum*, "Festschrift für Winternitz." 1892.

[2] *Matthes*, "Klinische Hydrotherapie." Jena, 1900, p. 226; ***Boas, Inte*** ***Rundschau***, 1894, Nr. 6.

In the treatment of all cases of gastroptosis the following must be remembered:

We may relieve and improve a state of insufficiency of the gastric musculature which develops in consequence of an unfavorable position of the stomach, and brings about severe nutritive disturbance in the patient. The low position of the pylorus, however, remains, and, as is the case with a damaged cardiac valve, only hypertrophy of the cardiac muscle produces compensation; in the same way greater labor is demanded of the musculature of the pylorus on account of its unfavorable effect upon the propulsion of the chyle. The efforts of the physician must be directed, on the one hand, to removing the disturbance of compensation which has occurred, and he must subsequently try to prevent such a condition. This part of his work is practical and of the utmost importance. An attempt should also be made to strengthen the abdominal walls and, in so far as possible, to prevent the patient from over-burdening his stomach. In how far this may be done in the individual case by giving minute directions in regard to the food can be ascertained only by careful observation.

The surgical treatment of gastroptosis rarely comes into question, because the continuance of the symptoms, notwithstanding careful treatment, must probably be attributed to a disturbance of the nervous system. Duret and Rowsing have proposed to attach the gastric wall to the anterior parietal peritoneum. Bier[1] advises us to shorten the gastrohepatic ligament by folding and stitching it, and in four cases he had favorable results by this means. The previous nutritive condition, which was very poor, the cachexia of gastroptosis, and the nervous symptoms were said to be relieved by this treatment.

DISPLACEMENT OF THE KIDNEYS—WANDERING KIDNEY—NEPHROPTOSIS

While displacement of the stomach almost invariably develops after puberty, displacement of the kidneys is often observed in children. This anomaly was even noted in a nursling.[2]

A male nursling, a few days after birth, was attacked by serious vomiting and symptoms of collapse. Physical examination revealed a small oval tumor at the right arch of the ribs which could be moved over the entire abdomen. The other kidney was also movable, but not to the same extent. The attacks recurred in the next few days, spasms simultaneously taking place. There was marked constipation.

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[1] *Bier, Zeitschr. f. Chirurgie*, 1901.

[2] *Rees Phillips, The Lancet*, 1903, p. 731.

A case of this kind shows that displacement of the kidneys
congenital. In by far the great majority of cases, however, it d
gradually, as is evident from the following statistics of L. Kuttne
The age in 326 cases was as follows:

1-10	10-20	20-30	30-40	40-50	50-60	60-70
6	32	82	123	49	26	8

The right kidney is most frequently displaced. According to
and Kuttner, we may assume that the right kidney is seven or eigh
more frequently displaced than the left kidney. Often the posi
both kidneys is changed, and in the majority of cases greater mo
may be determined in the right than in the left. E. Hahn[2] attribu
to the firm attachment of the left kidney to the descending colon (
little inclined to displacement) and to the pancreas and the spleen
the right kidney possesses but one point of fastening, the hepa
ligament. The sexes are affected by this anomaly in about the pro
of 7 or 8 to 1; among 97 cases of nephroptosis Landau[3] found
occur in men and 87 in women. Hahn, it is true, assumed that
kidney was 20 times more frequent in women than in men.

In regard to the absolute proportion of cases showing change
tion it is difficult to give a positive opinion. From investigations
living subject, Ewald and Kuttner assume 15 per cent., and with
reports of anatomists coincide. In the reports of the seventh de
the preceding century, among the autopsies of the pathological in
of Berlin, Vienna and Kiel, movability of the kidney was found
0.1 : 0.4 per cent., but the more recent investigations of Fischer-
in Kiel, show from 17 to 22 per cent.

According to the degree of movability of the kidney we diffe
various grades. Under physiologic conditions, the kidneys move
from their position with each respiration, descending on inspirati
rising on expiration. Some clinicians, such as Israel, Litten,
and Becher, maintain that this movability may be determined by p
even during life, while the majority of authors deny this. At all
therefore, in the normal person this movability must be very slight
the majority of healthy adults the kidney cannot be palpated f
abdomen. In the minority this is to some extent possible, but
lower third of the organ can be felt. Of course, thin, flaccid a

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[1] *L. Kuttner, Berl. klin. Wochenschr.*, 1890, p. 364; compare in the sa
Ewald's lecture before the Berlin Medical Society, March, 1890, and the
in connection with it. (Virchow, Litten, Landau, P. Guttmann, Sena
and others.)

[2] *E. Hahn, Zeitschr. f. Chirurgie*, LXVII, p. 356 *et seq.*

[3] *L. Landau*, "Die Wanderniere," Berlin, 1881.

walls will extraordinarily facilitate the examination. Becher and Lennhof[1] point to a special structure of the body which lends itself to palpation. Although tall persons usually form the bulk of this group, a definite bodily proportion is decisive, and this may be found in individual persons and in various races under different conditions. The greater the distance from the manubrium sterni to the symphysis pubis, the greater the portion of the kidney susceptible to palpation. If the slight physiologic movability of the kidney is increased, it is possible during palpation of the abdomen not only to feel a small portion of the kidney but two-thirds, or even the complete extent, of the organ; under the influence of the respiratory movements the displacement becomes particularly prominent. In a later stage of abnormal movability, the kidney can no longer be recognized in its normal position upon the posterior wall of the abdominal cavity, but may be felt in some other area of the abdomen, and thence it may either be readily moved or it becomes adherent to this region so that it can only be displaced in connection with other abdominal viscera.

ETIOLOGY

A primal cause of abnormal movability is probably a congenital predisposition. In the normal person the kidneys are usually situated in a groove in the posterior abdominal wall, anterior to and alongside the transverse processes of the vertebræ, and extending from about the height of the twelfth cervical to the third lumbar vertebra. Two Russian investigators, Wolkow and Delitzin,[2] have especially called attention to the importance of the formation of this paravertebral niche for holding the kidney. If this niche is markedly flattened, the kidney, naturally, will much more readily slip out. Besides this congenital flatness of the groove, which is readily seen, scoliosis of the vertebral column adds to the condition. We must also consider that in intra-uterine life the kidneys are low down in the abdominal cavity in the sacral hollow of the pelvis. Gradually they move up higher. This shows at once that the kidneys are not supplied with very tense suspensory ligaments; they are partially held by their ligamentous apparatus, but are also supported by the peritoneal layers and the fatty capsule; on the other hand, the kidneys maintain their position by the pressure of all the abdominal viscera. Accordingly, by a sundering of various links, this girdle which supports them may become slack or broken. A flattening of the niche simultaneously with a loosening of the ligamentous apparatus seldom causes a displacement of the kidney. Rigid abdominal walls, such as are found in most young,

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[1] *W. Becher* und *R. Lennhof*, *Deutsche med. Wochenschr.*, 1898, Nr. 32, und *Verh. d. XVIII. Congr. f. inn. Med.*, p. 476.

[2] *Wolkow* und *Delitzin*, "Die Wanderniere," Berlin, 1899; compare also *M. Zondek*, "Die Topographie der Niere," Berlin, 1903.

robust persons, usually hold the abdominal organs in their natural p
This explains the rarity of floating kidney in persons under twent
of age. When, from any circumstance, pressure in the abdomen
ened, this favorable influence is lacking. Lacing is the most f
among the deleterious factors which must here be considered, becs
liver is thereby pressed downward, and the right kidney, which
mately connected with the liver, also has pressure exerted upon it
explains the fact that the right kidney shows abnormal displacei
much more frequently than the left. We, however, are under
pression that lacing is here not so predisposing as is the case in
tosis. Repeated pregnancies which markedly diminish the support
abdominal wall mostly have a decided effect in that a portion of the
inal contents, usually the intestines, bulge forward and downward,
lateral pressure upon the kidneys is diminished.

The greatest decrease in pressure occurs when the abdomina
suddenly becomes smaller, as after the operative removal of large
Under such circumstances, Israel noted that, before his very e
kidneys became abnormally movable.

A gradual decrease in pressure may also be expected when from
emaciation considerable adipose tissue disappears from the ab
cavity. Other deleterious effects may also be operative, such as s
the abdominal wall. Thus I would explain the wandering kidne
I several times saw develop in emaciated patients in the course of
nary disease. In emphysema and asthma floating kidney may e
produced, because, from the emphysematously distended lung, pr
exerted upon the diaphragm, and then referred to the liver and t
kidney. Repeated shock to the abdominal wall from paroxysms
may gradually loosen the kidneys. In one case of wandering kidı
ing abdominal symptoms the patient attributed it to prolonged
In a similar manner straining during a difficult fecal evacuation, c
ing a heavy load, may be the cause. Under such circumstances t
origin of wandering kidney is quite possible, while in a normal, well
kidney this theory is scarcely plausible.

Just as the stomach may subsequently be displaced by a
which adds to its weight and dilates it, so may this condition
the kidney from hydronephrosis or pyonephrosis. Such cases,
are not wandering kidneys in the usual acceptation of the term.

SYMPTOMS

Symptoms of wandering kidney may be entirely absent. Th
in this instance, are usually strong, plethoric persons otherwise
whose kidneys, one or both, have descended into the abdome
causing any symptoms. Occasionally abnormal sensations are n

incidentally make known to the person in question that he has a movable kidney. Some patients report after unusual exertion a sensation of rumbling or one like the gliding about of a heavy substance. It is difficult to decide whether these reports depend merely upon auto-suggestion, or to what extent indefinable sensations, due to the influence of already existent factors, may subsequently be explained. It is still more difficult to assign vague nervous symptoms, whatever their nature, to nephroptosis. While we cannot doubt that all varieties of nervous symptoms may appear in persons whose kidneys are normally attached, yet, when people are predisposed to nervous affections, we are justified in assuming a movable kidney to be the cause of various painful sensations such as sciatica, intercostal neuralgia, or similar affections. Sensitiveness in the region of the iliac fossa may be attributed to the kidney. In any case the physician is wise not at once to communicate the fact that during an examination he has accidentally discovered a wandering kidney. The precautions yet to be considered, such as wearing a bandage, refraining from lacing, etc., can also be insisted upon when we speak to the patient merely of *"a predisposition to wandering kidney,"* and the first duty is to prevent the production of a floating kidney.

Symptoms on the part of the stomach are most frequently noticed, and these usually simulate a chronic gastric catarrh; acute attacks, designated as gastric spasm, are very rare. As a rule, we note loss of appetite, eructations, and pressure in the gastric region, less frequently vomiting. We know from numerous observations that in diseases of other organs, and particularly in those of the abdomen, the stomach is likely to become implicated. A heavily coated tongue is almost invariably present in disease of the gall-bladder, of the urinary bladder, and in inflammation of the renal pelvis, and the appetite is always decreased. It must be borne in mind that nephroptosis is often combined with gastroptosis. These patients are usually pale and weak young persons in whom gastric difficulties are prominent; stout, elderly women, even when a pendulous abdomen has developed, are more rarely affected. According to statistics, the implication of the stomach in nephroptosis is about as follows:

In 89 cases of movable kidney Kuttner found the position of the stomach normal in only 10, that is, the greater curvature of the stomach was situated normally in the median line between the umbilicus and the ensiform process.

In 79 cases the greater curvature was 3 to 4 cm. below the navel, occasionally even lower than this; in 15 of these cases, by inflation of the abdomen with air by means of a double bellows, a true prolapse of the stomach could be observed.

Dyspeptic symptoms were present in 70 of these persons, hence this symptom may be designated as the most important and most common in floating kidney.

A peculiarity of the gastric symptoms in nephroptosis is their increase after muscular exertion and their disappearance, or at least improvement, after prolonged rest. As a rule, the majority of these female patients suffer most during the menstrual period.

In nephroptosis, just as in gastroptosis, constipation is important and is due to the same causes—weakness of the abdominal walls, insufficient nourishment or food poor in residue, often kinking of the colon, etc.

Symptoms on the part of the kidneys are rare. It might readily be supposed, and would seem likely, that torsion and kinking of the ureters would occur frequently. As a matter of fact such an accident is very rare. Dittl, Landau, Lindner and others have, it is true, described attacks which resembled renal colic and which were mistaken for this. Occasionally it was believed that spasmodic contraction of the ureter could be felt through the abdominal walls. Some patients are attacked so suddenly with sensations of nausea, vomiting, and severe pain in the renal region, the ureters and the bladder, that the physician must assume severe peritoneal irritation, for the pulse also is small and increased, the skin is cool, cold sweat appears, etc. But such intermediate attacks which probably depend upon kinking of the ureter or of the vessels, or torsion of the renal nerves, are rare; I have only once seen a case of the kind. Albuminuria and hematuria are infrequent, as are also so-called intermittent hydronephrosis, pyonephrosis, and pyelitis. Up to the present time but little attention has been given to the latter complication, therefore I shall briefly relate my own experience in regard to it. [These attacks are the so-called "Dittl's crises."]

Pyelitis as a Complication of Hydronephrosis.—The patients, usually women about forty years of age, who for ten or even twenty years have had no symptoms of their wandering kidney, are suddenly attacked with acute pains in the abdomen. The pains, as shown by palpation of the abdomen, are chiefly located in the displaced kidney, and radiate along the course of the ureter to the region of the bladder. Strangury frequently occurs. There is fever from 100.4° to 102.2° F. which is remittent in type. The urine is turbid, usually of acid reaction, and microscopically is composed almost exclusively of pus corpuscles. Chemical examination, of course, reveals albumin, but decided amounts (up to 5 per 1,000) are also found in the filtered urine or in the clear urine after sedimentation has occurred.

After one or two weeks the inflammatory symptoms subside. The temperature becomes normal, but the pulse for some time continues to be accelerated. The sensitiveness of the kidney gradually passes away. The albumin decreases so rapidly that after two or three weeks none can be detected in a clear layer of urine; but the excretion of pus and the turbidity of the urine due to this may continue for months and, occasionally, even for years. For a long time the patients are able to take but little exercise and must avoid all exertion. Prolonged sitting is impossible. A slight tenderness of the urinary organs upon pressure continues to exist for some time, and makes the wearing of bandages or corsets irksome. *Slow recovery from pyelitis and floating kidney is characteristic.*

In the treatment warm drinks, warm baths, hot poultices, etc., are most effective; among drugs salol, urotropin, uva ursi and the like.

In one case, eight days after a very movable right kidney had become affected I saw the left kidney also attacked, this, so far as could be ascertained, being in its normal situation. The fever, which had declined, again rose, and all of the symptoms returned.

The sudden appearance of pyelitis without other symptoms may be attributed to an acute infection; but the nature of the infection is not clear. If gonorrhea or cystitis have not previously been present, it is very likely due to an emigration of bacteria from the intestine (Posner). But such complications are rare, for Kuttner saw only 4 among his 89 cases, and among 93 cases operated upon by Küster[1] only two displaced kidneys showed the development of pathologic processes.

DIAGNOSIS

The diagnosis of floating kidney is in some cases very easy. Even upon superficial palpation of the abdomen, we feel an organ of the size and consistence of the kidney which either moves about or is fixed in some area. In the majority of cases the organ is situated upon the right side of the body, which confirms the opinion that the right kidney is by far the most frequently displaced. The diagnosis becomes difficult when the kidneys still retain their position in the posterior abdominal wall. Bimanual examination then becomes necessary. As already stated, only in those persons who are tall and thin is it possible to feel a portion of the normally situated kidney, that is, about one-third of the organ, and this is most readily perceptible in the deeper situated right kidney. To overcome the marked tension of the abdominal walls which is often present, Becher and Lennhof advise an occasional examination in a warm bath. A simpler method which I found serviceable was the employment of a moist, hot poultice about the abdomen.[2]

For the examination, the patient is placed in a horizontal position, the legs are flexed somewhat, and abducted and rotated outwardly to ensure a comfortable position, then one hand is placed upon the back at the

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[1] *P. Geis*, "18 Jahre Nierenchirurgie," Marburg, 1900.

[2] The relaxation of the tension of the abdominal walls is naturally also of value in the consideration of other pathologic processes. For diagnostic purposes in perityphlitis, the occasional employment of the hydropathic poultice to determine the presence and extension of the exudate is especially useful. The enlarged appendix can then occasionally be palpated.

[illegible] gradually [illegible] of fat, [illegible] examinati [illegible] a doubtful [illegible] we may draw [illegible] under normal circum [illegible] method of exa [illegible] on the dark

[illegible] the findings, we must first [illegible] congenital change, a so-c [illegible] taught us by his specia [illegible] upon an embryonal [illegible] and upon the back) must be made, the alteration in position of the movable organ must be followed and investigated, and we must observe whether it is connected with other organs. According to J. Israel,[1] the proof that the kidneys are out of their normal positions is alone decisive, since constriction of the right lobe of the liver,[2] carcinomatous and tubercular tumors of the ascending colon and of the flexure of the colon, carcinomata of the pylorus, small ovárian tumors with long pedicles, and enlarged gall-bladder have been mistaken for floating kidney.

(For confusion with wandering spleen see page 293.)

TREATMENT

The treatment of floating kidney must be limited in most cases to the application of a suitable bandage, to preventing the formation of a pendulous belly, and, in case this is already present, preventing its further enlargement. It is easy to understand that the symptoms of wandering kidney are not so readily removed by a bandage as those of gastroptosis. If pyelitis has preceded, such a bandage is often intolerable to the patient.

Occasionally a corset specially designed is useful, yet often this fails to have any effect. Then we are compelled to resort to strips of adhesive plaster. Rose[3] advises the following:

"Three strips as wide as the hand and of varying length are used, the longest being applied firmly around the belly; the entire abdominal mass is firmly pressed upward from below, and the two ends of the adhesive strip are brought together at or over the vertebral column. The crest of the ilium should remain free, but the plaster must be applied immediately above, and adjacent to, this bone. The propping of the abdominal wall is strengthened by the application of two additional lateral strips which also meet at the vertebral column, running upward and backward from Poupart's ligament."

If these bandages do not at once relieve the discomfort of the patient, we should insist upon a few weeks of rest in bed.

Plentiful nourishment and prolonged bodily rest will, as a rule, remove the symptoms which appear in very nervous patients who suffer from floating kidney. By this treatment fat will perhaps be deposited in he belly and the capsules of the kidneys, and thus a cause of abnormal movability will be removed. The nervous symptoms, too, are usually re-eved by a rest cure in suitable environment and by the employment of ydrotherapeutic measures. But, in the end, the results do not entirely correspond with our hopes. After a short time, the old complaints wandering kidney are again heard.

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[1] *J. Israel*, "Chirurgische Klinik der Nierenkrankheiten." Berlin, 1901, p. 20.

[2] *Penzoldt*, *Münchener med. Wochenschr.*, 1903, Nr. 10.

[3] *Rose*, *Zeitschr. f. prakt. Aerzte*, Sept., 1901; quoted from *B. Presch*, "Die physi- isch-diätetische Therapie in der ärztlichen Praxis." Würzburg, 1903, p. 170.

The inefficacy of treatment indicates that operation for the re of the symptoms should be considered. The excision of the kidne been proposed and carried out, but, fortunately, this is no longer ad by any surgeon. The knowledge that when one kidney is very mo the other organ often shows the same disability, although to a less e should have prevented such a method of procedure. Even when the c tion of suture, yet to be touched upon, would be useless, extirpation be rejected, for, no matter how great the nervous symptoms, the important disease is in the nervous system, so that, as a rule, the re of the kidney brings no permanent improvement. The dangers o operation may best be seen from E. Hahn's[1] report of 42 extirpatio wandering kidney, with the result that 11 patients succumbed to peritonitis and uremia.

The operation usually employed at the present time is suture kidney to the abdominal wall (nephrorrhaphy), introduced by E. The kidney is exposed by excision in the lumbar region, the fatty is partly removed and fastened to the muscles by a few stitches t the capsula propria and the substance of the kidney. According to under some circumstances, the repetition of nephrorrhaphy, perh another method, may be necessary. Objections have been raised operation. In the first place, Hahn himself emphasizes that it quite devoid of danger. The mortality is said to be from 2 to 3 p Furthermore, the cure is often not permanent, the difficulties b fact, not alone due to the kidneys, but also to a simultaneously gastroptosis, and symptoms referable to the stomach and to the system are combined. According to the reports of various surge cures may be estimated at 50 to 60 per cent. From this the co is obvious that the operation is advisable only in such cases of kidney as present also renal symptoms, therefore, symptoms of tion, of hydronephrosis or pyonephrosis, or a stubborn, distressing This, as has been stated, is only the case in a minority of the p

DISPLACEMENT OF THE LIVER WANDERING LIV (HEPATOPTOSIS)

Displacement of the liver is comparatively rare, provided the complete descent of this organ from its usual position. [illegible] grees of displacement, however, are comparatively frequent, as shown when [illegible] lying [illegible] [illegible] found in who have borne children. When the liver descends into the lower abdominal cavity, we naturally first attribute it to an abnormal weakness of the suspensory ligaments, and, secondly, repeated pregnancies which weaken the abdominal walls are frequently held responsible. Thus Landau [1] found a very marked displacement of the liver into the lower abdominal cavity in a woman, aged 28, who in three years had passed through four pregnancies.

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[1] [illegible] 1883.

Cantani [2] was the first to describe such a change of position, and other cases were subsequently reported by various authors. In 1885, Landau published a comprehensive monograph, "Ueber Wanderleber und Hängebauch," in which these alterations in the position of the liver are accurately described as well as their mechanical effect upon the position of the abdominal organs.

Among 31 cases of floating liver which Landau collected from literature, 27 occurred in women and 4 in men; 14 of these cases were his own patients, and he expressly points out that most authors underestimate the frequency of wandering liver because the liver is rarely examined while the patient is in the erect posture. The age of the person affected is usually over forty years.

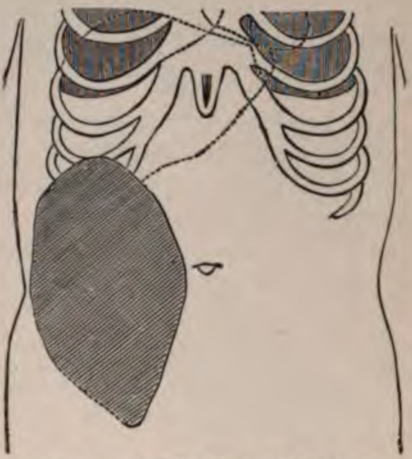

FIG. 16.—Displacement of the liver.

Hepatoptosis is recognized by the absence of liver dulness in the usual area, and by the discovery of a movable tumor of the size and consistence of the liver in the lower abdominal cavity; movability generally occurs when the body changes its position. In many cases, displacement of other organs can also be determined; Landau, for instance, among his 14 cases of wandering liver found a floating kidney in 4.

The position of the liver is best shown by the accompanying illustrations taken from Landau's book.

Confusion with cancer of the stomach, tumors of the omentum, and ovarian cysts may occur if we do not bear in mind displacement of the liver, and thus fail to trace the boundaries of the liver in the right hypochondrium.

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[1] *L. Landau*, "Die Wanderleber und der Hängebauch der Frauen," Berlin, 1885, comprehensive compilation of the literature.

[2] *Cantani, Schmidt'sche Jahrbücher*, CXLI.

SYMPTOMS

Symptoms of wandering liver are usually manifest, for the organ is so large that, when in an unusual position, its pressure upon other organs could not be unnoticed. A sensation of fulness, rumbling in the abdomen, ill-defined pains which sometimes extend over the entire abdomen, to the chest, to the small of the back, and to the right shoulder are, according to Landau, the most frequent signs. As in displacement of other organs, the patients complain most after exertion; on prolonged rest in the recumbent posture the symptoms disappear. Some patients are annoyed by the pulsation of the abdominal aorta and other large abdominal vessels which is distinctly felt. Sometimes it is impossible to trace an intimate connection between the nervous symptoms and the abdomen. Disturbances of gastric digestion and of bowel action are often noted, as in displacement of all other abdominal organs.

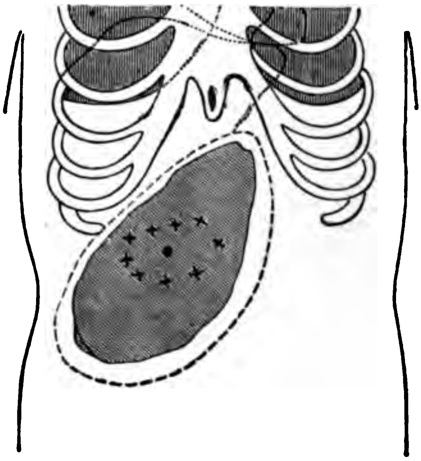

FIG. 17.—Displacement of the liver.

TREATMENT

The treatment generally consists in applying a binder to support the pendulous abdomen. Schott[1] has constructed a shield resembling the human hand to hold the liver in its normal position, but, like all other apparatus to support the abdomen, the results are not very satisfactory.

DISPLACEMENT OF THE SPLEEN—WANDERING SPLEEN

A displacement of the unenlarged, normal spleen is very rare. Glénard reports that he found it only twice in 160 cases of enteroptosis. The location of the spleen near the diaphragm is such that in all pathologic processes which dilate the left thoracic cavity it is forced downward, and thereby is more readily reached by the palpating finger; but there is no greater permanent movability than in the normal organ. A displacement of the spleen is also observed when it becomes enlarged by pathologic processes (by uncompensated valvular lesions, hepatic cirrhosis, malaria, leukemia). But, even in this case, the movability is usually not great. Finally, those cases remain in which extreme movability of the spleen is noted in some area of the abdominal cavity, most frequently in the left iliac fossa. The cause of this displacement is unknown. Flaccidity of the abdominal walls or shock naturally favors its occurrence; but only in a very slight number of persons, and these mostly women, are the ligaments of the spleen so feebly developed as to permit a marked displacement.

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[1] *Schott,* "D. Medicinalzeitg." 1882, Nr. 21 und 22.

DIAGNOSIS

In the diagnosis, a differentiation must be made between wandering spleen and the far more frequent floating kidney. The spleen frequently shows upon its anterior border one or more incisures which are lacking in the kidney. The entire configuration of the spleen is different from that of the kidney. It must be remembered, too, that the right kidney is most likely to be displaced, and this is generally detected, although not always, upon the right side of the abdomen. When the spleen is absent from its normal area on percussion the diagnosis is certain, but this is a proof naturally very difficult to obtain in the obese and in very old persons. The kidneys should either be palpable in their normal position, or their absence decided upon. In conclusion, they may be confounded with very movable ovarian cysts having long pedicles.

SYMPTOMS

Symptoms of wandering spleen may be entirely absent; occasionally there are pains of very indefinite nature. The turning of the pedicle and consequent compression of the splenic vessels may cause atrophy or even gangrene in the surroundings of the organ or in the stomach. In one case the spleen steadily decreases in size, the disturbances gradually disappear, and recovery takes place; in another, peritoneal processes may develop. But these are probably great rarities, always current in medical literature.[1]

TREATMENT

In the treatment, the employment of an abdominal bandage must first be considered; then, by the administration of arsenic, quinin, and iodin, we must attempt to decrease the size of the spleen; finally, the nervous symptoms should be combated by hydrotherapy.

Among surgical measures, fixation of the spleen, splenopexy, which was first proposed by Rydygier[2] is prominent. Litten[3] quite properly raises to this the objection that permanent fastening of an organ ' to so great an extent takes part in the respiratory excursus of th phragm can hardly be expected. When the spleen is decidedly enl however, its total removal may be considered. It is self-evident tha is by no means a simple operation[1] and that it should never be a when nervous symptoms only are present.

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[1] It is quite remarkable how few reports there are in the literature of the last few decades regarding wandering spleen, which is a proof of the rarity of the affection, for the obverse conclusion that everything relating to this condition has been ascertained, and that, therefore, further reports would be superfluous, is unjustifiable.

[2] *Rydygier, Verhandl. der deutschen Gesellschaft f. Chirurgie,* Berlin, 1895.

[3] *Litten,* "Die Krankheiten der Milz," Wien, 1898, p. 37.

DISPLACEMENT OF THE COLON (COLOPTOSIS)

In 1853 Virchow[2] called attention to the fact that displacem the colon was of frequent occurrence, and attributed this to local c peritonitis and to acute, infectious, intestinal diseases, especially tery. Displacement of the colon is due to the same causes as g splanchnoptosis, to a weakness of the ligamentous apparatus and cidedly pendulous abdomen. The greatest deviation in course is by the transverse colon, particularly by the right flexure, which oc ally forms a sharp angle whose vertex is situated below the um the entire large intestine resembling in shape the letter M. Under circumstances, particularly in aged persons, portions of the in packed with feces are felt in the right iliac fossa, a finding wh me to regard perityphlitis as the consequence of fecal stasis, wher now know with certainty that the feces above the ileo-cecal valve a fluid in composition, the solidification chiefly taking place in the verse colon.

Displacement of the colon may be proven by inflation from the r Such a process, however, is usually superfluous, because valueless in ment. For although displacement of the colon may hinder the prop of the intestinal contents, as has been stated, an obstruction in the tinal canal is easily compensated for by a greater activity producing trophy of the smooth musculature of the intestine. The causes of pation may be readily found in weakness of the abdominal muscl the composition of the food which leaves too little residue, and often gastric disturbances. Nervous symptoms may usually be attributed general splanchnoptosis and to neurasthenia rather than to coloptosi

The most effective treatment in displacement of the colon as w any other part of the intestines consists in the application of a well- abdominal binder, and the regulation of the diet and the entire m life, in the manner already described.

Just as in gastroptosis, Bier attempted the surgical treatment of sis. In a patient who suffered from constipation accompanied by pain the left side of the abdomen, the mesocolon of the transverse colon, hich was extremely tortuous, was straightened by silk sutures.

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[1] *Compare Vulpius, Beiträge zur klin. Chirurgie*, XI, 1894; *Kirchhoff, Ther Monatsh.*, 1898.

[2] *Virchow's Archiv*, V, 1853.

[3] *Blocher, Zeitschr. f. Chirurgie*, LVI.

From the description just given, which shows that the symptoms deend only in small part upon displacement of the colon, it is evident that uch surgical treatment is not advisable.

DISPLACEMENT OF THE HEART—WANDERING HEART COR MOBILE[1]

Under physiologic conditions the heart is held in position by the aorta and the pulmonary artery which are firmly adherent to the pericardium, and also by its attachment to the diaphragm in the thorax; it is still further supported by the large veins which empty into the heart. As is shown by the illustration (Fig. 18), taken from Determann's[2] work, the heart is attached at such points on its right and upper side as will permit to the apex of the heart the greatest possible movability in conformity with the movements of the thorax, and this position is most distinctly revealed by the apex beat. As is the case with the abdominal organs, the heart is also maintained in position by the pressure of the adjacent organs. Thus the lungs completely surround the heart and exert pressure which, in the case of pleural effusion or any other process which decreases the space in the thorax, may increase to such an extent as to force the heart away from its normal position. The heart is also held in place by its attachment to the diaphragm, and particularly to the central tendon, by means of which every variation in pressure within the abdominal cavity is transmitted to the heart (see Fig. 19, taken from C. Toldt's[3] *Anatomical Atlas*).

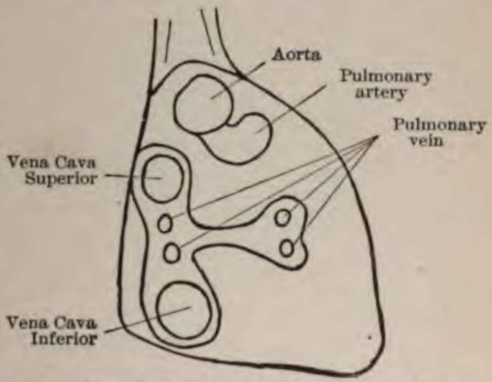

FIG. 18.—Pericardium with vessels. (After Henke.)

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[1] The term employed by some authors, *cardioptosis*, does not appear to me to be appily chosen, in which opinion A. Hoffmann coincides, since it is too suggestive of low position of the cardia, therefore of a portion of the stomach.

[2] *Determann, Deutsche med. Wochenschr.*, 1900, Nr. 5; compare also the discusion following this.

[3] *C. Toldt, Anatom. Atlas*, III, Aufl., IV, Liefer., p. 482, Berlin und Wien., 1903.

To a certain degree every heart is movable; this has been known to clinicians ever since the introduction of percussion. It has also been known that the heart could be displaced 1 or 2 cm. to the left, and, to a somewhat slighter extent, also to the right. But the investigations of Rumpff, Determann, A. Hoffmann, Cherchewski,[1] L. Braun, Leusser and others have given us more accurate information. Determann, in particular, deserves great credit for having widened our knowledge of this process by a large number of researches, in which the results of percussion were subjected to a subsequent test by X-rays. These results, later confirmed by other investigations, are as follows:

In a normal person in the left lateral position displacement of the heart takes place, upon the average 2 to 3 cm. to the left, and in the right lateral position about 1 cm. A displacement is rarely absent, but occasionally it may be very slight. In some healthy persons, however, this displacement is decided, and amounts to 6 cm. to the left and 4 cm. to the right. Generally, these extreme degrees of displacement are found in middle-aged persons who are poorly nourished and have a weak muscular system, in whom, therefore, an abnormally great displacement of the abdominal organs is also most frequently found. This coincides with the opinion that in women the heart is generally much more movable than in men. In the newborn, cardiac displacement is almost unknown, and in children it is very slight. A deviation from the normal state of the abdominal viscera is found when, in the aged, the movability of the heart does not increase but decreases, probably because, by augmentation of the emphysema, the pressure exerted by the lungs upon the heart becomes greater. But the pressure from the abdominal cavity influences the movability of the heart still more than pressure from the lungs. During pregnancy the heart is forced upward, and rests upon the diaphragm in such a way that hypertrophy is simulated; yet the heart is but slightly movable. Soon after labor, however, the highest grades of cardiac displacement may be noted, as a rule, to the left and upward, so that the apex beat appears near the axillary cavity. A similar influence is exerted by all other processes in the abdomen, such as ascites, meteorism, by a pendulous belly, or enteroptosis.

Emaciation, as a rule, intensifies the movability. In the first place, the layers of fat normally surrounding the heart and which reach their greatest development (Gerhardt) in the obese occasionally disappear; the

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[1] *Cherchewski, Gaz. méd. de Paris*, 1887, No. 53; *Rumpff, Verhandl. d. VI. Congresses f. innere Med.*, 1888; *Deutsche med. Wochenschr.*, 1902, Nr. 31, und 1903, Nr. 3; *A. Hoffmann*, also, 1900, Nr. 19; *L. Braun*, "Ueber Herzbewegung und Herzstoss," Jena, 1898 u. *Centralbl. f. innere Med.*, 1902, Nr. 35; *Leusser, Münchener med. Wochenschrift*, 1902, Nr. 26; *Pick. Wiener klin. Wochenschr.*, 1889, p. 747; *A. Schmidt, Deutsche med. Wochenschr.*, 1901, Nr. 16. I have also made investigations in a small number of cases.

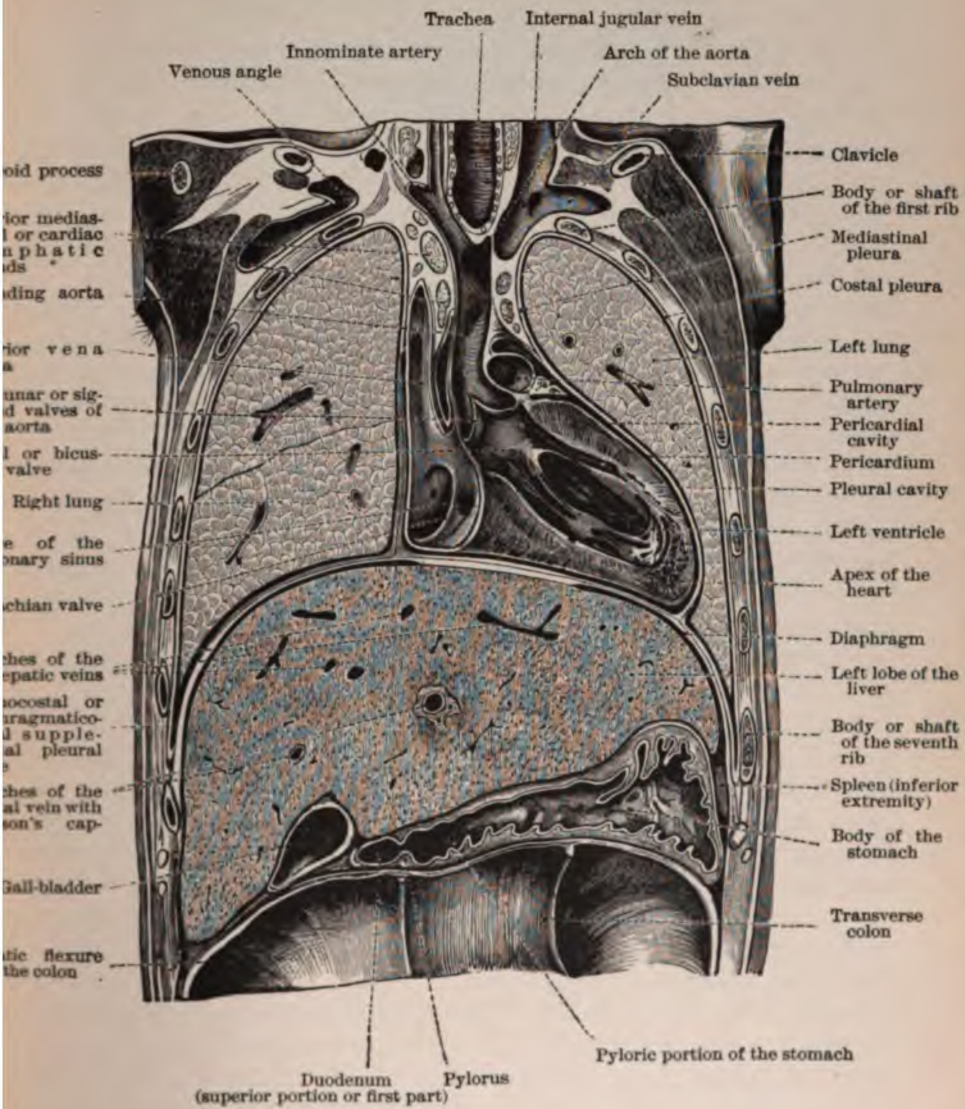

Fig. 19.—Frontal section through the trunk.

abdominal fat is largely decreased also in hyponutrition. This, there favors a loosening of the viscera from their attachments, a down displacement of the diaphragm, and, thereby, leads to a decrease of sure in the thorax.

Movability of the abdominal organs and of the heart is also obs in chlorosis. Hoffmann considers this the explanation of the p dilatation after exertion so frequently reported by some investig (probably from erroneous observations), but he is probably incorrect. as we know that the chlorotic has a flaccid muscular system, and under these circumstances a displaced stomach readily dilates, so the may be assumed of the heart. In this case we would have a complic of abnormal displacement and dilatation.

We have little definite knowledge of the pathologic processes are influenced by the amount of space in the thorax and abdominal c Some clinicians (Curschmann, Braun, and Cherchewski) maintain, their experience, that arterio-sclerosis particularly involving the aor creases movability, while Determann was unable to confirm this.

It is easily understood that just as a neoplasm burdens the sto or hydronephrosis produces a tendency to displacement, so the conditions arise in a hypertrophied heart. An enlargement of th ventricle may become so extensive that dilatation of the right ver escapes observation (Fränkel). Individual influences play a great and often a hypertrophied heart shows no displacement.

As a rule, displacement is readily proven by palpation and percu

SYMPTOMS

As to the symptoms, there is no unanimity among clinicians. such as Rumpff and Leusser, incline to the view that certain sym distinctly indicate cardiac displacement, wandering heart. Persons marked displacement of the heart cannot sleep for any length of upon the left side without distress arising. Symptoms become notic such as palpitation, oppression, difficulty in respiration, attacks of sy fear, irregularity of the pulse, etc. Other clinicians, such as Brau Romberg, deny this, and quite properly emphasize that many person a greater or less displacement of the heart without any symptoms. over, the persons who usually suffer from wandering heart are excee nervous, and often complications are present, such as alcoholism, c asthenia, etc., to which the symptoms are attributed; this makes an o exceedingly difficult.

In the main, I agree with the latter view, and in this connec wish to reiterate what I have said regarding floating kidney. A h person will usually feel no discomfort from the displacement of a or of the heart, although it cannot be denied that displacement of organs, when made known to him, and particularly at the onset, may give rise to symptoms. The rarity of cardiac symptoms in healthy women after labor, during which the heart is especially prone to be displaced, favors this view. Moreover, I recently saw a marked displacement of the heart during an obesity cure, while, simultaneously, the symptoms referable to it disappeared. The case was that of a woman, aged 32, having a height of 1.7 meters, and weighing 98 kilograms; the obesity had particularly increased during the last few years. The heart at first revealed normal dulness, the pulse during rest in the sitting posture was from 80 to 88, but upon the slightest exertion it rose to 130, and at the same time decided dyspnea appeared. Questioning elicited the report that, for many years, the patient had slept only upon her back or in the right lateral position. I instituted a moderate hyponutrition, and assured the lady that after the loss of 16½ to 22 pounds she would be able to sleep upon the left side. When, after six weeks, she had lost 20 pounds, quite a decided displacement of the heart to the left could be determined, about 4 to 5 cm., which had not previously been noticeable. Of course, I did not tell her this. General bodily activity, as well as that of the heart, had increased, the pulse during rest did not exceed 80, and even upon exertion dyspnea did not so rapidly appear. Above all, the lady assured me that she could now sleep the entire night upon her left side without any inconvenience.

Here, therefore, the antifat cure produced such an improvement in the circulation that possible abnormal sensations due to displacement of the heart were unnoticed by the patient.

In this respect erroneous opinions may easily be formed because undernutrition is not well borne by nervous patients. In men who work under mental strain, and in women who have many household cares, it is apt to produce nervous disturbances, while, after they are relieved from their burdens and pressing duties, and are sent away to a pleasant environment, most of them bear a loss in weight of from 11 to 16½ pounds without the consequent displacement of the heart having any unpleasant effect.

Upon the basis of these views, the removal of the symptoms of wandering heart by plentiful nutrition might be supposed the correct treatment. But, generally speaking, this is not the case. We must not forget that in some persons blood formation is immoderately increased by plentiful nutrition, and thus higher blood pressure arises. We do not know under what circumstances cardiac hypertrophy occurs, but it must all the more be reckoned with since the development of this affection usually escapes observation. It is quite likely that disturbances regarded as "cardiac neuroses" mark its onset, usually designated cardiac hypertrophy following "luxury consumption." In practice, even after minute investigation of the heart, we may sometimes doubt whether certain symptoms are due to an increased displacement or to a beginning hypertrophy. Only in thin, debilitated individuals does hypernutrition appear to be justi The efforts of the physician must be chiefly directed to combating nervous symptoms by means of rest, a proper regulation of the musc activity, plentiful amounts of fresh air, and, perhaps, also the employ of hydrotherapeutic measures. In some cases the binder advised by in Nauheim, a so-called heart brace or heart bandage, may be adv The chief value of the apparatus is, however, merely suggestive; a events, unlike the advantage from the employment of the binder in placement of the abdominal organs, it is impossible to explain by p mechanical laws the benefits from its use.

DISEASES OF THE PANCREAS

SYMPTOMATOLOGY OF THE DISEASES OF THE PANCREAS

By L. OSER, Vienna

HISTORY

Although physiology and experimental pathology were busy in the last third of the preceding century with a study of the pancreas which has led to a better understanding of the various vital functions of this organ, practical medicine has utilized this knowledge to but very slight extent.

Hospitals and medical colleges, especially the departments of surgery, have devoted increasing attention to the pancreas within the last few years, and it has come to pass that, in making a diagnosis of digestive disturbances from certain alterations of metabolism, the pancreas also has been considered, yet in the popular current of professional life, diseases of the pancreas have been almost ignored.

Regrettable as this apparent apathy is, its cause is easily understood when sought for. Chief among the reasons is the fact that, until recently, this indifference was manifest also in the schools, in the clinics, and in the laboratories. Even to-day, adequate attention is not devoted to the pancreas, as is evident when we consider the manifold and complicated questions brought up for consideration, questions which often relate to elementary but most significant processes. Even pathologic anatomy has added little to our knowledge of the pancreas; at autopsies, the pancreas frequently is not considered at all, or but superficially investigated, and a cursory examination of this organ is usually without value. It is true the difficulties are great. An apparently normal pancreas will, upon careful microscopic investigation, frequently show changes which are exceedingly difficult to interpret correctly for the reason that the organ, during the death agony and, perhaps, also for some time after death, undergoes change from continued self-digestion, and this may very readily be mistaken for a pathologic alteration which had occurred during life.

In the last few years a decided change for the better has taken place, as is shown by the increasingly rich literature. Since the important researches of v. Mering and Minkowski, which point with certainty to the connection between diabetes and the pancreas, an active interest in this organ has been awakened and, owing to the victorious advance of su following the initiative of Gussenbauer, the pancreas has becom border-land between internal medicine and surgery, and incontrove proof has been furnished of the great practical importance of this

The pancreas to-day, on account of individual causative observa is not only the subject of discussion in inaugural dissertations, but p nent exponents of theoretical and practical medicine in different cou have devoted their best endeavors to the development of a positive fc tion for the study of diseases of the pancreas. Naturally we are st from the goal—but a few important facts have been ascertained, whi not only interesting to the practical physician, but the knowledge of he cannot and dare not ignore in his professional work. This justification for calling close attention to the present status of c knowledge concerning diseases of the pancreas.

This article will be devoted to a description of the *symptoms* by we are enabled to make a diagnosis of disease of the pancreas.

ANATOMY AND PHYSIOLOGY

Before entering upon my actual theme, I desire to emphasize points in the anatomy and physiology of the pancreas which have s bearing on our subject. First in importance, the gland, as a ru *two ducts*, a fact which is not always borne in mind, even by physic since in the animal usually experimented upon—the dog—two du invariably present. The tying of *one* duct, or the introduction of nula into *one* duct, may lead to error. In the dog—as recently r by Helly—these ducts are always distinctly separated, which in the exception. Helly, under the direction of Zuckerkandl, has studied this subject thoroughly. He examined 50 cases, and amo he only once found the ductus Santorini and no ductus Wirsungia all of the other cases he found the ductus Santorini connected w ductus Wirsungianus, or the duct existed alone, the latter conditic much the rarer. Of the 50 cases, Santorini's duct had free pass the intestine in 40 cases, and was obliterated in 10.

The relations between the pancreas and the ductus choledochu the utmost practical importance; the latter enters a groove upon of the gland, bends toward the duodenum, and, as Zuckerkandl embeds itself, but soon terminates in a canal. Helly, in his inve of 70 cases, confirmed these reports of Zuckerkandl. He found terminal portion of the ductus choledochus is always closely c with the head of the pancreas to the extent of from 2 to 7 cm.

Pawlow made some interesting experiments in regard to inn According to this author, the vagus is the secretory nerve of the but this nerve also carries fibers which have an inhibitive secretc tion; in the sympathetic Pawlow also determines secretory and vasoconstrictory fibers. More recent investigations by Wertheimer and Lepage have demonstrated that, after the severing of all nerve centers, secretion does not cease; that, therefore, there must also be an automatic nerve apparatus present in the gland. These investigations permit us to conclude with certainty that in pathologic cases disturbances in secretion may also be caused by disturbances of innervation.

The incessant labors of physiologists in the last few decades have frequently demonstrated the great importance of the physiological functions of the pancreas. Undoubtedly the pancreas is one of the most important organs of the body; in digestion and in metabolism perhaps the most important. The pancreatic juice is the only glandular secretion which is capable of converting all substances that can be at all digested into that form which is necessary for their complete utilization as food. It is more active than any other ferment (trypsin) in the splitting of the albumin bodies; it emulsifies and splits the fats so that they are capable of absorption (steapsin); it transforms the carbohydrates into sugar (pancreatic diastase) and also possesses a milk-coagulating ferment. The secretions of the other digestive glands also possess these properties, but they are not combined in the secretion of any *single* organ. The specific property of the pancreas, which belongs to it alone, is the *splitting up of fat.* In the intestine, it is true, a certain degree of fat-splitting is possible, but only by bacteria. The enormous importance of the pancreatic juice in digestion has been decisively proven by animal experiments, to which we shall later revert. But its *digestive* function is, however, not the only activity of the gland. It also performs an important part in the human economy by the transformation and preparation of sugar. We attribute this function to the *internal secretion,* the conclusive, experimental proofs of which we shall later discuss minutely.

This sketch indicates the great importance of the pancreas in the human economy. Disease of this organ must, naturally, produce severe disturbances, and we might suppose that, in consequence, such marked symptoms would appear that the recognition of a disturbance in function would present no difficulty, but the contrary is the case. How can this fact be explained? The most significant reason is this—that only very extensive or complete destruction of the organ and the occlusion of both ducts will cause characteristic symptoms. In partial disease, or the occlusion of *one* duct, as we shall see, the internal as well as the digestive function may remain intact, and the normal remainder of the gland, although with only *one* duct, and perhaps vicariously, may compensate for the functions of the neighboring organs. Total or very extensive disease is certainly much rarer than partial, and the latter usually gives rise only to doubtful symptoms which are but uncertain points of support for its recognition. The symptoms are interesting, because, as a rule, not only the pancreas that is diseased, but a simultaneous disease o neighboring organs may be the cause, or consequence, of disease o pancreas, and its physical signs be much more distinctly and obvi apparent than those of the diseased pancreas. If, for example, ch pancreatitis develops in the course of gall-stone disease, cholelithiasis i the most prominent symptom, and the signs of disease of the pan may be completely disguised.

The **symptoms of absence of function,** therefore, are of paran importance in diagnosis, and to these we shall devote our attention.

SYMPTOMS

First among these are *diabetes* and *alimentary glycosuria.*

Autopsy findings long ago directed attention to the relation bet the pancreas and diabetes. The oldest, most reliable report was in by Cowley, who, in a diabetic, found numerous calculi in the subs of the pancreas. Chopart later made a similar observation. Brigh 1833, saw, in a diabetic, aged 19, a hard, nodular tumor at the he the pancreas while the gland itself was atrophic. Since that time, si cases have been frequently mentioned in literature.

The frequent coincidence of diabetes and disease of the pancreas c Frerichs, Seegen, and Friedreich to assume a causal connection betwe clinical and anatomical findings; but it was still questionable wl disease of the pancreas was the cause or the consequence of di French authors—above all, Bouchardat—took a more decided stand proposed a clinical type, diabète maigre, and looked for its foundat a disease of the pancreas. This assumption was enticing, and wa frequently confirmed by conclusive reports of cases; nevertheless, mained but a hypothesis until by animal experiment v. Mering anc kowski finally furnished the incontestable proof that by removing stroying the pancreas in a dog severe diabetes occurred. A radical in opinion was the result, and it is now believed beyond all doul diabetes in man is caused by disease of the pancreas. The results ol by these authors were soon confirmed by many others, and in an series of animals diabetes was produced by extirpating the pancrea diabetes occurred only by removal of the *entire* gland, or, at least *greater portion*; *partial extirpation* gave varying results. If one- one-fourth of the gland were allowed to remain, v. Mering and Mi frequently found no glycosuria. But severe diabetes might also duced. This depended particularly upon the composition of the of the gland remaining. If this was subsequently destroyed by tive inflammation or atrophy, diabetes developed; cases of dial medium severity, or only a transitory or more or less permanent ali cosuria arose; or, as already mentioned, in the great majority of cases form of sugar excretion took place. Sandmeyer found, in a patient whom severe diabetes developed 13½ months after partial extirpation f the pancreas, that diabetes persisted for eight months and until death ccurred. In the experiments in partial extirpation conducted by Katz nd myself, it was impossible to produce severe diabetes, but only *transitory* and *alimentary glycosuria*; in some cases, sugar was absent from the urine.

Minkowski definitely proved that the cause of diabetes in extirpation of the pancreas could be found neither in a lesion of the nerves nor in injuries, and that it could not be attributed to the absence of the pancreatic secretion.

This was demonstrated in an indisputable manner by the transplantation of portions of the pancreas under the abdominal skin, first by Minkowski and later by Hédon. A portion of the pancreas may be detached and, without injuring the vessels which supply it, may be transplanted under the skin of the abdomen. If the portion remaining in the abdominal cavity is removed, no glycosuria occurs, not even the alimentary form; if the section underneath the abdominal skin is subsequently removed, severe diabetes is produced. If the transplanted portion is badly nourished, mild diabetes may occur. If complete atrophy takes place in the grafted portion, severe diabetes develops. The mere tying of the ducts never leads to diabetes, except when such a change due to these processes subsequently occurs in the glandular substance that it is completely destroyed.

It is absolutely true that removal of the pancreas in quite a number of animals has produced diabetes. Besides its digestive function, the pancreas unquestionably has another which is related to sugar metabolism. We do not err when we refer this function to a secretion quite distinct from the digestive juice; this is called the *internal secretion,* or, following Hansemann, the positive function. In what manner this internal secretion exerts its activity is still doubtful. Minkowski, and also most authors, assume that the pancreas in the preparation of sugar also generates a product which acts in the organs.

A series of hypotheses have been proposed in explanation, but no definite proof of the correctness of one or the other is yet at hand.

The hypothesis of Lépine is most interesting; he assumes a glycolitic ferment which is normally produced in the pancreas. This glycolitic ferment reaches the lymph, thence the blood, particularly the white blood-corpuscles, and carries on the preparation of sugar in the tissues. If this ferment is absent, hyperglycemia and diabetes result. Lépine later modified this hypothesis, no longer referring the point of attack of the glycolitic ferment to the blood but to the tissues. In consequence, as Minkowski points out, the assumption of such a ferment is no longer impossible, lthough we have no actual proof.

Other hypotheses, such as those of Chauveau and Kaufmann, in wl an increase of sugar formation in the liver is assumed to be the b and also the views of the brothers Cavazzani are disproven by the ex ments of Minkowski, who demonstrated that in experimental pancre diabetes not an increase of the sugar production, but a decrease in consumption, occurs. We only know, therefore, that the pancreas h function which regulates the consumption of sugar, but we do not k the nature of this function.

An attempt has lately been made to discover the region in which internal secretion is found. Langerhans in 1869 described structur the pancreas which differ from the pancreatic tubules. These so-c intertubular cell clumps are also found in man and in various ani and have different forms. Recently the property of furnishing the int secretion has been ascribed to these intertubular cell clumps. Lag as well as Diamare some years ago expressed the view that these clumps bear some relation to the internal secretion.

Recently Walter Schulze has attempted to solve this question e mentally. He ligated small portions of the pancreas in guinea pigs studied the changes which followed. After a few days an atrophic p developed in the glandular elements which continually increased— after 80 days, at which time the tubular area was completely atro being replaced by delicate connective tissue; the cell clumps had no part in this atrophy, but, on the contrary, showed not the sli change. Schulze concludes from this that these cell clumps are subst structures independent of the glandular system of the pancreas. true this does not prove that they have any bearing on sugar metab It had long been believed that these structures were in their nature di from the secreting glandular elements. Langerhans himself regarded as nervous elements; other authors have looked upon them as lym tissue. Some authorities believe them to be embryonal remains, an have also been considered in connection with the secretion of the di ferment.

Walter Schulze's experiments are evidently of importance, b proof that these structures do not belong to the glandular system a related to the internal secretion must be furnished by further investi Szobolew recently undertook to prove by chemical and microscopi vestigations that Langerhans's cell clumps have some relation to metabolism, and decided this to be a fact. In two cases of pancrea betes the disappearance of these cell clumps was confirmed. Opie at the same conclusion; in 11 cases of interlobular pancreatitis in form of the disease Langerhans's cell clumps are visible only w process is very far advanced, he noted mild diabetes only once, and in a case in which the sclerosis had also attacked Langerhans's cell In three cases of interacinous pancreatitis in which form also the ously mentioned cell clumps were implicated, diabetes was present twice, and in the fourth case of diabetes there was complete hyaline degeneration of Langerhans's islands.

If we succeed in proving positively that these cell clumps are concerned in the internal secretion, important knowledge as to the true cause of pancreatic diabetes will have been gained.

The demonstration in animals of an experimental pancreatic diabetes enables us to understand much more fully than was previously possible the numerous autopsy reports in literature of changes in the pancreas in the diabetes of man. All doubts disappear, and it is certain that in a large number of cases disease of the pancreas was the cause of the diabetes. At the necropsy of diabetics various changes were found in the pancreas, most frequently atrophy, but often also induration, calculi formation, carcinoma and fatty degeneration. Doubtless, some of these changes in the pancreas are not the cause of diabetes; in many cases they are either its consequences or are in no way related to it. Atrophy has often been demonstrated to be the cause of the marasmus which occurs in diabetes. Hansemann reports a characteristic form of *diabetic atrophy* with well developed symptoms of an active process added to the process of the secretory cells; this belongs to the varieties of interstitial inflammation, similar to that in certain forms of granular atrophy of the kidneys. Hansemann describes early stages of these processes, and designates them as anatomical individualities which must necessarily lead to diabetes.

If, in many cases of diabetes, no changes are found in the pancreas, not even upon minute microscopic examination, this is additional proof that there are other causes of diabetes than disease of the pancreas—a view now generally accepted. Lanceraux in the last Congress at Paris maintained the unity of diabetes, and of its origin in disease of the pancreas. He assumes that there are anatomical determinable changes or functional disturbances of the pancreas which lead to diabetes. Lanceraux formulates a hypothesis against which weighty objections may be raised, and which cannot be sustained at this present time.

A vulnerable point in the theory of pancreatic diabetes in man is the fact that *total destruction* of the pancreas has been found without diabetes having existed during life. Hansemann's hypothesis does not sufficiently explain the fact that in cases of total destruction of the pancreas by diffuse carcinoma, carcinomatously degenerated cells may perhaps furnish the internal secretion of the pancreas, as there are also other varieties of total destruction of the pancreas without diabetes. Perhaps future investigation of the anatomical findings revealed in Walter Schulze's experiments will clear up the situation. No matter what the conclusion may be, this loop-hole cannot alter the fact that in man diabetes is due to the absence of the pancreatic function.

Not only permanent diabetes but also *transitory and alimentary glyco-*

suria may be caused by changes in the pancreas; this is shown by animal experiments, as well as by the facts demonstrated in the disease in man. After various injuries of the pancreas, even although slight, transitory glycosuria may occur. In 15 out of 32 cases of partial extirpation, Minkowski saw transitory glycosuria. Minkowski, as well as myself, demonstrated alimentary glycosuria in the previously mentioned experiments. In one of these there was a transitory excretion of sugar after every operation upon the pancreas. Upon an exclusive meat diet, on the administration of 10 grams of grape sugar slight glycosuria occurred, and after 50 grams it was marked. In an interesting article Wille has furnished proof that alimentary glycosuria in man is also connected with changes in the pancreas. In 800 patients in the Hamburg General Hospital, Wille made feeding experiments, administering 100 grams of grape sugar in each case, and, with a positive result, this was repeated once weekly. In 47 cases he found alimentary glycosuria. In 77 of the 800 cases an autopsy was held; in 15 of these alimentary glycosuria had been recognized during life, and at the autopsy high-graded changes in the pancreas were invariably found. Ten doubtful cases still remained in which during life alimentary glycosuria had been proven, and at the autopsy marked changes in the pancreas were found. Alteration in the pancreas was also observed at the autopsy in cases which, during life, had given no evidence of alimentary glycosuria. Wille arrived at the conclusion that the regular appearance of alimentary glycosuria is an important diagnostic sign of existing disease of the pancreas, that in a periodically appearing alimentary glycosuria the suspicion of disease of the pancreas should not be at once rejected, but that, in the absence of this symptom, the pancreas cannot be looked upon with certainty as normal.

These reports of Wille are noteworthy; they show that cases of *constant alimentary glycosuria* may be the result of changes in the pancreas. But the relatively small number of positive reports prevents our proving with certainty that this is always the case. Alimentary glycosuria—also the constant form—is frequently found associated with various diseases, particularly those of the nervous system. In these numerous cases but few autopsy reports relating minutely to the pancreas are at hand, and it may be assumed that there are cases of alimentary glycosuria not caused by changes in the pancreas. It may be true, as in permanent diabetes, that constant alimentary glycosuria occurs without changes in the pancreas. In spite of these objections, we may maintain, on the basis of our present knowledge, that the proof of permanent diabetes as well as of constant alimentary glycosuria justifies us in including changes of the pancreas in our diagnostic calculations.

A second group of symptoms develops from the absence or by the destruction of the *digestive function.* We know that the pancreas has fat-emulsifying and fat-splitting, a proteolytic and an amylolytic, function. Disease of the pancreas or its destruction must necessarily lead to disturbances of this kind. The fact is also operative that a large portion of the gland, or almost all of it, must be incapable of performing its function, hence symptoms due to the absence of function must appear. Add to this the circumstance that, as a rule, two ducts are affected, in some animals even more, and it becomes clear that if only *one* duct be occluded, provided that in the other the secretion is sufficiently active, no digestive disturbances may follow. This explains why we so rarely observe disturbances of digestion as a result of disease of the pancreas.

Let us first consider the absence, or the disturbance, of *fat digestion.* This is shown either macroscopically, or only microscopically or chemically in the changed condition of the feces.

For a long time isolated clinical observations have pointed out the connection between impaired assimilation of fat and changes in the pancreas, and an attempt was made to prove this experimentally in animals. Even Claude Bernard was of the opinion that pancreatic juice splits the neutral fats, emulsifies them, and is therefore necessary for the absorption of fats. Among those who differed with him was Schiff. Opinions varied until the discovery of experimental pancreatic diabetes by v. Mering and Minkowski, and the digestive disturbances following the removal of the pancreas were studied. Although even to-day there are diversities of opinion and many doubts concerning these points, nevertheless the view that, in the absence of the pancreatic function, disturbances arise in the absorption of fat is generally accepted. Abelmann, a pupil of Minkowski, found that with an absence of the pancreas non-emulsified fat is not absorbed at all, and emulsified fat only to a slight extent; only in the case of cream was the absorption more complete. With partial extirpation, 50 per cent. of emulsified fats was absorbed and 80 per cent. of milk. Upon the addition of pancreas to the food, digestion improved. Fat-splitting, even with a total absence of the pancreas, was undisturbed. Similar reports were made by Sandmeyer, Cavazzani, and Baldi. Very different results were obtained by Hédon and Ville who raised objections to Abelmann's method of research, and in this they were supported by Pflüger. Hédon and Ville found that after total extirpation of the pancreas, fat absorption continued, although to a lessened extent; in a case of severe diabetes it amounted to about 18 per cent.; fat-splitting remained about normal, principally free fatty acids, but also small amounts of soap. In more extensive investigations Siegfried Rosenberg ascertained that in experimental, gradual destruction of the parenchyma of the gland, fat absorption still showed almost normal values; that, however, upon increasing glandular destruction, it is damaged. In regard to fat-splitting, which Rosenberg attributes mainly to bacterial action, he believes that the low fat-splitting figures found with increasing glandular destruction are due to the frequent evacuations, and are caused by the undigested mat which passes through the intestine.

In complete unanimity with the results of animal experiments, cli experience teaches us that frequently, but by no means always, in di of the pancreas, in chronic inflammation, in the case of neoplasm cysts, of stones in the ducts, and in atrophy, a deficient digestion o can be proven. The first undoubted report is by Kunzmann in 1 he saw a profuse discharge of fat in the feces of a man who suffered induration of the pancreas with obliteration of Wirsung's duct, ch jaundice and dropsy. A report by Fles is very interesting. A dia who had eaten considerable bacon and fat meat discharged ounces o in his feces that could be separated from the stool. If the patient with his food an emulsion prepared from the pancreas of a calf, th disappeared. At the autopsy high-graded atrophy of the pancreas found. In a case of carcinoma of the head of the pancreas observe me, there were present for months uncommonly copious, thick, p fecal discharges which were always profusely admixed with fat. In cases jaundice was absent—a fact which must be particularly empha for, according to the excellent researches of Friedrich Müller, if th is absent from the intestine fatty stools are quite normal.

The forms of disturbance of fat digestion which are combined disease of the pancreas vary considerably. They are sometimes reco macroscopically, and are then designated as *true steatorrhea.* In thi dition large quantities of oily or fluid, yellow and yellowish-brown, masses are discharged with the evacuations, or even independent of In the cold these become compact, resembling butter, grease or wa hardened fat may then completely envelop the feces contained wit Přibram, in a case observed by him, closely analyzed these fatty disc and proved their similarity to the varieties of fat ingested. Ch examination of the fatty masses which are visible to the naked eye neutral fats and fatty acids.

In diseases of the pancreas the stools are of the consistency o grayish-white, or colorless, occasionally asbestos-like, and the in amount of fat can only be recognized by chemical and micros processes. Unquestionably there are many transitional stages betwe steatorrhea and the previously mentioned *fatty stools,* so that the di between the two forms of dejecta may be assumed to be a graduat The chemical investigation of these feces chiefly reveals neutral fat acids and soaps. Such dejecta, rich in fat, are found by no mean sively in diseases of the pancreas, but may also occur normally in who have eaten such a great amount of fat that the intestine is in of absorbing it; in the majority of cases, however, the condition is c when bile is absent from the intestine, in certain diseases of the i in. amyloidosis, in tuberculosis of the intestine, in distributed atr the mucous membrane of the small intestine, in caseation of the mesenteric glands, in chronic tubercular peritonitis, and perhaps—to which also Nothnagel calls attention—in intense catarrhal processes. It is evident from this fact that a high percentage of fat in the stools does not prove disease of the pancreas, and we must first exclude all other causes of insufficient fat absorption before we can regard an affection of the pancreas as the cause of the disturbed fat digestion.

Müller, who was positively unable to determine a greater amount of fat in the stool upon the absence of pancreatic juice from the intestine, designated *diminished fat-splitting* as a factor of disease of the pancreas. Müller excised the pancreas of a dog, and, under antiseptic precautions, placed it in sterilized milk in the incubator for 24 hours. Fatty acids amounting to from 42 to 45 per cent. were present. In a control experiment in which the pancreatic ferment was destroyed by boiling, no fat-splitting occurred. After Müller had previously proven by experiment that intestinal bacteria had but a slight influence upon fat-splitting, he concluded that the important factor for fat-splitting in the intestine is contained in the pancreatic juice, and the absence of the pancreatic function is expressed by disturbed fat-splitting. In a series of cases of pancreatic disease, Müller proved the disturbed fat-splitting with certainty, his reports being confirmed by several authors, but denied by others.

The question has been most thoroughly studied by Katz. He formulated an expeditious method which enables us to determine the degree of fat-splitting in 24 hours. In the examination of a number of cases observed in my hospital, in which during life reduced fat-splitting, and at the autopsy pathologic changes in the pancreas, were determined, he came to the conclusion that a diminution of fatty acids and soaps to below 70 per cent. of the total amount of fat in the fatty stool favors a diminished or even completely arrested function of the pancreatic juice; but in nurslings, and in profuse diarrhea, this decrease had no significance. Other authors have also noted disturbed fat-splitting in pancreatic disease, for example, v. Noorden, Rosenheim, Anschütz.

Diminished fat-splitting in disturbed pancreatic function may, therefore, be considered as proven, and the validity of this proof does not suffer from the fact that there are undoubted cases—perhaps many—in which no disturbance of fat-splitting can be determined; even in widely distributed disease of the organ, a healthy portion of the gland may serve the excretory ducts, which may discharge a sufficiency of pancreatic juice into the intestine.

Where a disturbance of fat-splitting has been found without disease of the pancreas being determined, the circumstances are different. Katz specified that in nurslings and in profuse diarrhea reduced fat-splitting might be found without coexisting disease of the pancreas. Certainly in all cases in which an increased peristalsis rapidly propels the contents through the intestine, the pancreatic juice, normal in amount, will no have sufficient time to split the fat to such an extent as is possible unde normal circumstances.

Zoja, who recently published a comprehensive study of fat absorption, mentions two cases of reduced fat-splitting without changes being found in the pancreas or in the excretory ducts. In one case there was stenosis of the large intestine, and in the other carcinoma of the gall-bladder. Zoja recognizes in deranged fat digestion an important indication of an affection of the pancreas, but, on account of the great number of reported cases he arrived at the conclusion that the disturbed fat-splitting is not the most important symptom, but the small amount of soaps which he invariably demonstrated in diseases of the pancreas. Deucher had previously called attention to the importance of this factor. The demonstration in the feces of a decided quantity of neutral fats, or of a great amount of fatty acids, particularly, however, of a deficiency in soaps, according to Zoja, points with great likelihood, if not with positive certainty, to the absence of pancreatic juice from the intestine. The main stress is placed by Zoja upon the lessened quantity of soaps. The greater the percentage of soaps, the more certainly may an occlusion of the pancreatic ducts be excluded; the less the quantity of soaps in proportion to the neutral fats and fatty acids, the more readily may occlusion of the pancreatic duct be assumed. The less the amount of pancreatic juice which flows into the intestine, the slighter is the amount of alkali, and, therefore, also the smaller the amount of soaps. In this reasoning Zoja finds no difference between the animal experiment and observation in man, which has also been emphasized from the standpoint of fat-splitting, since Abelmann, as well as Rosenberg, found smaller amounts of soaps.

We cannot enter here upon a criticism of the work of Zoja. I shall only mention that Abelmann reported the frequent finding of quantities of soaps which reached the normal. Müller previously objected to this method of determining soaps and the combined fatty acids, and designates the proportion of free fatty acids to the soaps in the feces as an inconstant one which depends upon accidental conditions. I must also state that in a case of Zoja's of recognized pancreatic affection, normal values for soaps were found. The relative value of the factor emphasized by Zoja, the lessened soap figures in proportion to the large amount of neutral acids and fatty acids, cannot be denied; but more complete investigations are necessary finally to solve the important question of the diagnosis of diseases of the pancreas. We can only state positively that disturbed fat digestion is an important symptom in pancreatic disease. An increased amount o fat in the stools is alone not a basis for the assumption of a pancreati affection. If no jaundice exists, and no disease of the intestine is presen if increased peristalsis by which the ingesta are rapidly propelled throug the intestine does not explain the insufficient fat digestion, then the su icion of disease of the pancreas is certainly justified. Some forms of ltered fat digestion, true steatorrhea, in which larger, even macroscopically recognizable, amounts of fat are passed with or without the stool, which show diminished fat-splitting with a certain increased amount of at in the stools, and low quantities of soaps in proportion to the fatty cids and neutral fats, are factors which point with great likelihood to isease of the pancreas. It can only be positively proven, however, when ther symptoms due to absence of function, such as diabetes or insufficient itrogen absorption, or certain clinical symptoms, such as tumor, bronzing of the skin or pancreatic colic, are present.

Equally important is the *absence or disturbance of albumin digestion,* which is observed experimentally and clinically in diseases of the pancreas. Abelmann found that when the pancreatic juice is absent, only a portion of the proteids is absorbed; this averages 44 per cent. in animals in whom the pancreas was removed, and 54 per cent. in the cases of partial extirpation of the pancreas. If, on meat diet, pig's pancreas was simultaneously given, the absorption of nitrogen was decidedly increased, and 74 to 78 per cent. was absorbed. After a meat diet the presence of numerous macroscopically recognizable undigested muscle fibers was conspicuous. Similar results were obtained by de Renzi, Cavazzani, Sandmeyer, and Harley. In an instance of total extirpation of the pancreas performed by Katz and myself, and which was followed by diabetes, after meat was taken for the first time, large portions of undigested meat were macroscopically found in the feces. Under microscopic investigation numerous muscle fibers with distinct transverse striæ were detected.

Rosenberg's investigations were most thorough; after tying the excretory ducts, injecting into them an acid solution, and subsequently tying again, as is done for the gradual destruction of the glandular parenchyma, he found at once that the absorption of nitrogen was but slightly altered, even before he could determine any disturbance in the absorption of fat and carbohydrates. Later, after the removal of the degenerated glands, nitrogen metabolism fell to 33 and 35 per cent., while 41 per cent. of fat and 50 per cent. of carbohydrates were still being absorbed.

Quite as positive is the clinical proof based upon numerous observations that in man disease of the pancreas causes disturbances of albumin digestion. Deficient meat digestion is most conspicuous. In the feces there are numerous undigested muscular fibers, as may sometimes be recognized with the naked eye, but microscopic examination shows them very distinctly. In the case of a diabetic, reported by Fles, the dejecta contained uncommonly numerous, undigested, quite distinct, transverse muscular fibers. If the patient took calf's pancreas with his other food, the meat was again perfectly digested, and the muscle fibers again appeared in the stool when no pancreas was added to the food. Harley recognized a similar condition in a case of pancreatic abscess. v. Ackeren noted impaired digestion of meat in carcinoma, Küster in a case of cyst, and Lich in a case of calculus formation in the pancreas. In a case of carci of the pancreas that came under my observation, I noted numerous re of transverse, striped, muscular fibers with distinct muscular stru Zoja recently reported a case of carcinoma of the head of the pa with occlusion of Wirsung's duct and of the common gall-duct, in numerous fibers of meat were found in the feces with distinctly oped transverse striation. A similar condition was observed by Aue in a case of carcinoma of the pancreas, and by Cipriani and Giudicea in pancreatic calculi.

The insufficient nitrogen absorption in pancreatic disease has been proven by exact researches in metabolism. Hirschfeld conduc series of thorough analyses in diabetes. He found that 32 per ce the nitrogen substance ingested could be found in the feces, while 5 per cent. corresponds to the norm. It is true this was not prov autopsy findings. But this proof was present in a case of Weintr 45.2 per cent. of the albumin of the food, and 22.2 per cent. of th were lost. At the autopsy a markedly contracted, indurated pancreas decided increase of the connective tissue was found, and in this onl lated glandular globules could be seen. Weintraud, in agreement wit results of Rosenberg's animal experiments, lays the principal stress the fact that in diseases of the pancreas proteid absorption is mo cidedly, or at least as greatly, disturbed as fat absorption; wh intestinal diseases, in which the intestinal apparatus for the absorpt fat functionates insufficiently, as in amyloid disease of the intestine the absorption of fat is comparatively as much affected as that albumin. The conditions were similar in a previously mentione reported by Zoja, of carcinoma of the pancreas with occlusion of Wi duct. More than 70 per cent. of the nitrogen of the food was excrete hardly 30 per cent. absorbed. Unfortunately, but few reports of ments are at hand; but even with the data we have, there can be no that this is a positive method for the recognition of diseases pancreas.

Recently Sahli has proposed another method for testing the di function of the pancreas for proteids. He determined by exact expe that gelatin capsules hardened with formaldehyd—he calls them capsules—after reaching a definite degree of hardness, are soluble by water nor by hydrochloric acid pepsin digestion, but only so by atic juice (by trypsin). If these capsules are filled with a substanc can be recognized in the saliva or in the urine—best with iodofo cases of arrested pancreatic digestion a reaction for iodin does no at all, or only very late. With normal motility of the stomach ar ciently good pancreatic function the iodin reaction in the saliv itself in from 4 to 8 hours. According to Sahli, from the absence the delayed appearance of the iodin reaction we may determine a disturbance of the pancreatic function, provided the motility of the stomach is not reduced. These reports of Sahli are very interesting, and if, in a large number of tests, the absence or the late appearance of the "glutoid reaction" coincides with anatomical proof of a pancreatic disease and arrested secretion, we have a positive and infallible sign of disease of the pancreas.

There can be no doubt that the *digestion of carbohydrates* suffers when the pancreatic function is absent or disturbed. Rosenberg has considered this in his animal experiments. So long as the pathologic process in the pancreas is not too far advanced, normal or almost normal absorption was found; when the process had made further progress, the figures decreased decidedly, so that, in this case, digestion was already seriously damaged. In an experiment at the conclusion of which the degenerate glandular remains were removed, carbohydrate absorption as well as fat-splitting sank rapidly and decidedly, with a loss in the absorption of nitrogen and fat; on pancreas feeding, in all the experiments a conspicuous improvement was noted. Clinical reports in regard to this factor are very few. In recent isolated cases, as, for example, by Auerbach and Edoardo Italia, it is expressly stated that considerable starch was found in the stools.

When the pancreatic function is either absent or disturbed, impaired digestion occasionally produces conspicuous symptoms that are often noted by the patient himself. The stools have a *massive appearance* which is out of proportion to the amount of food ingested. I have seen a number of cases in which this symptom led me to suspect a disease of the pancreas, and the further course of the affection justified this assumption. Much of the food passes through the digestive tract without being utilized. *In spite of plentiful food, there is constant loss of weight, and conspicuously massive, thick, pappy or compact dejecta, whose amount appears to be decidedly greater than the ingested food, and this is an important symptom which calls for the closest attention of the physician.* Exact microscopic and chemical analysis explains this massiveness; muscle fiber as well as fat and carbohydrates are excreted without being assimilated.

The symptoms due to absence of function unquestionably furnish the most important aid in the recognition of an affection of the pancreas, and must therefore be first described. There are, however, a few symptoms which cannot be regarded as denoting absence of function, which, nevertheless, enable us to recognize that the pancreas is diseased. Among these we must consider the following: *Bronzing of the skin, peculiar colic and pains of a definite character in the epigastrium; tumor or resistance in this region, and certain forms of jaundice.* These signs, with the exception of tumor formation, only become important when they are combined with permanent or transitory symptoms of absence of function of the gland.

French authors were the first to direct attention to a peculiar of diabetes with deposition of pigment in the skin—diabète bronzé. pigment formation is the expression of hemochromatosis, which dev in the skin as well as in the different organs. The color of the sk brownish, brownish-black, or somewhat grayish, the discoloration is *dif over the entire body,* and *nowhere are pigmented areas observed, not upon the mucous membrane,* and this differentiates the condition Addison's disease.

The cases reported up to the present time are few in number. I book I published in 1898 I mentioned 22, Anschütz, in 1899, 24 In most instances changes were found in the pancreas. It was scle rusty-brown, the excretory ducts were patulous. Jeanselme had al declared diabetes to be due to sclerosis of the pancreas which dev as a result of deposition of pigment. Anschütz recently studie undoubted case. During life, besides the symptoms of diabetes, there also signs of disturbed secretory function: distinct fat-splitting and stools. For this reason a diagnosis of disease of the pancreas was At the autopsy, a chronic, indurated pancreatitis with general hemoch tosis was found. The deposition of pigment caused chronic inflamm of the pancreas, and this in turn the diabetes. A similar report explanation were furnished by Opie.

Bronzing of the skin diffusely distributed, from which hemochrom may be concluded, absence of pigment areas in the skin and in the m membranes, and simultaneously diabetes, warrant the assumption o ease of the pancreas, particularly if, at the same time, secretory di ances, fatty stools, deficient digestion of fat and albumin can be mined.

From the *pain* alone—no matter what form it may assume—dise the pancreas can never be diagnosticated. In pancreatic affection may be of many varieties and degrees, it may be *persistent* or *parox* The *former,* sometimes remittent or paroxysmal, or gradually bec more severe, is found in abscess, in hemorrhage, in acute and c pancreatitis, and in tumor. In carcinoma the pain may, under so cumstances, be of extraordinary intensity, so that the patient an avoids any movement, shrinks at a loud word, and rejects foo combination with a feeling of extreme weakness and marked prost this pain shows certain peculiarities such as are very rare in other of the abdomen. Whether pressure upon the celiac ganglion or tor the same causes this peculiar pain has not been proven, but it is tionably a characteristic feature, and we are justified in entertain suspicion that the seat of the affection is the pancreas.

The *paroxysmal pains* may resemble sharp colic limited chiefly epigastrium, and, under certain circumstances, may also point to of the pancreas. By some authors special stress is laid upon th

Epigastric colic, as such, naturally proves nothing, not even that, for example, as in the case described by Münnich and Holzmann, its main point of concentration is under the left arch of the ribs. We know that renal colic or gall-stone colic, colic of the appendix, and a beginning pericolitis may cause similar pains in the region of the splenic flexure. We are, then, only justified in diagnosticating a disease of the pancreas if pancreatic concrements are found in the feces, or if, after prolongation of the disease, symptoms due to absence of function appear, such as diabetes, or insufficient digestion of proteid or fat. We may conclude from this that the same process that gives rise to prolonged colic also brings about changes in the parenchyma of the pancreas, and gradually disturbs or arrests the function of the organ. In calculi formation there are chronic inflammatory changes with connective tissue proliferation, and these gradually displace and replace the gland substance.

Not only in stone formation does colic occur, but also in all cases of occlusion of the excretory ducts and of the secretory passages in the interior of the gland, as from cicatrices, neoplasms, indurated inflammations or hemorrhages. In cysts of the pancreas these colics play a particularly important rôle. In 104 cases operated upon Takayasu found colic a symptom in 64 cases. As this form of pain is evidently very much more rare in other cysts of the abdomen, its presence, as Leube emphasized, is an important sign of pancreatic cysts. It may be asserted that these colics are much more frequent than is to-day assumed. How many so-called nervous gastralgias may depend upon pancreatic colic! Minute investigations in metabolism and absorption may, perhaps, show transitory or permanent anomalies which occur in the course of a pancreatic affection.

A *feeling of resistance or a tumor* in the region of the pancreas forms a significant symptom. Naturally we must be certain that this resistance, or the tumor, actually springs from the pancreas. This proof, on account of the concealed position of the organ, is difficult to establish. Primarily we must consider carcinoma, cysts, and indurative inflammation, as the acute processes, acute hemorrhagic pancreatitis, abscess, hemorrhage and necrosis are evidently much rarer occurrences.

In a small number of cases of *carcinoma,* in about ⅕ to ¼, a tumor can be felt, but even then the differentiation from carcinoma of the duodenum, from carcinoma of the choledochus and of the porta hepatis, from lymph-gland tumor, from aneurysms of the hepatic artery, from products of inflammation of a tuberculous nature, even from carcinoma of the colon or pylorus, is very difficult, occasionally impossible. The differentiation from tumors of the colon or pylorus is most readily made since pancreatic carcinomata are, as a rule, fixed, while carcinomata of the colon and pylorus are frequently movable. By artificial inflation of the stomach and colon, by the demonstration of symptoms of stenosis in the stomach or

intestine, by the chemical and microscopic investigation of the gastri
intestinal contents, as well as by other clinical signs referable to d
of the stomach, the intestine, or of the pancreas, the diagnosis may
sionally be made with certainty. Much more difficult, almost impo
is the differentiation of tumors of the duodenum or the biliary pas
Here the appearance of symptoms which indicate absence of functi
the pancreas may clear the situation.

The appearance of a *cystic tumor* in the region of the pancreas
not at all prove that we are dealing with a pancreatic cyst.

Echinococcus of the liver, of the spleen, of the mesentery and c
peritoneum, hydronephrosis, dropsy of the gall-bladder, cysts of the
tum or the mesentery, the spleen, the kidney or the adrenals, sof
comata of the liver, accumulations of fluid in the bursa omentalis,
may give rise to confusion. It would lead us too far afield to enter
the details of the diagnostic factors; frequently the diagnosis can o
made with certainty at the operation.

The *inflammatory tumor* is rarely recognized during life; usua
is confounded with neoplasms. Often, however, at the operation it
quite clear whether an indurative inflammation or a neoplasm is pr
and only from the gradual disappearance of the tumor, as, for ins
after the operative removal of gall-stones, do we recognize that a c
inflammation of the pancreas was present, and this appears to be a
means rare occurrence.

One of the most frequent symptoms of disease of the panc
jaundice. The intimate relations existing between the head of th
creas and the common gall-duct sufficiently explain this. Unfortu
there is no characteristic form of jaundice which indicates disease
pancreas. The form in which the jaundice slowly but steadily in
and finally leads to complete occlusion by compression of the c
gall-duct, is also found in tumors of the duodenum, of the hilus
and in lymph-gland tumors, all of these causing a similar devel
of the jaundice as well as the Bard-Pic syndrome: Intense, chronic
ually increasing jaundice with enormous dilatation of the gall-
rapid emaciation and cachexia with usually subnormal temperatu
the absence of decided enlargement of the liver are not confirmato
although it cannot be denied that this symptom-complex occurs m
quently in carcinoma of the head of the pancreas than in tumor
neighboring organs, or in stone formation in the biliary passage
relatively rare appearance of the cardinal symptoms, of diabetes
sufficient fat and albumin digestion, may, it is true, here also indi
correct diagnosis.

Much less convincing are other symptoms which are observed
ease of the pancreas: *Emaciation, digestive disturbances, salivation
ing, constipation, hemorrhagic stools, intestinal occlusion, fever.* I

a proof of a sequestrating pancreas, or of pancreatic concrements in the stools, may undoubtedly lead to the diagnosis of disease of the organ.

The acute inflammations, hemorrhagic pancreatitis, pancreatic apoplexy and necrosis continually appear with severe symptoms: Sudden, severe and paroxysmal pain in the gastric and umbilical region, nausea, vomiting, immediate and severe collapse, great anxiety, enormous increase in the pulse rate, and not infrequently the picture of acute intestinal occlusion. The differential diagnosis between internal incarceration and the previously mentioned processes is exceedingly difficult, and usually can only be made at the operation or at the autopsy.

In disease of the pancreas some authors lay special stress upon certain changes in the urine which enable us to make a diagnosis of involvement of the pancreas, such as an *insufficient excretion of indican* and the proof of *pentosuria* or *maltosuria*; unfortunately, these views have not been confirmed. During pancreatic digestion, as is well known, indol, the mother substance of indican, develops. For this reason we formerly, with a suspicion of disease of the pancreas, first directed particular attention to the urine. In a case of occlusion of the small intestine Gerhardi concluded from the fact that indican was not increased that disease of the pancreas was the cause of the occlusion; the autopsy confirmed Gerhardi's view. The animal experiments of Pisenti appear to confirm this opinion. In our animal experiments, at my suggestion, my assistant, Dr. Katz, invariably examined the urine for indican and always found this substance increased. Reports are at hand showing that, in the course of disease of the pancreas in man, the amount of indican was also increased.

Just as little has the appearance of *maltose* or *pentose* in the urine proven itself characteristic of pancreatic disease.

In reviewing the important symptoms which have been mentioned in this article, it is apparent that the most reliable aids in the diagnosis are the *symptoms due to absence of function of the gland, and the proof of resistance or of a tumor having its seat in the pancreas.* In a detailed description of the individual forms of the disease, we are convinced by the careful consideration and investigation of the different symptoms in a number of cases that the diagnosis may be made with certainty, or with more or less likelihood. In many—probably in the great majority—of cases, if we go beyond an assumption we find ourselves upon the wrong track. If any result be obtained it is only possible by persistent, tireless investigation. Such a result is not only of theoretic, but also of great practical value, as has been shown by the efforts of surgeons, who, interfering at the right time, have cured many affections which had endangered life, such as chronic and acute inflammatory processes, and neoplasms of the pancreas.

We are now at the dawn of rational treatment of disease of the pancreas, and undoubtedly internal therapy will also produce practical results when we are able to diagnosticate the by no means rare and curable aff tions of the pancreas. On account of the intimate relation between t biliary passages, the liver, and intestines on the one hand, and the p creas on the other, the consecutive changes in the pancreas will questionably—I might say will unwittingly—be cured by internal tre ment. When we gain a deeper insight into the pathology of the pancre and are able to recognize clearly the pathologic changes in this organ will perhaps often be possible not only to prevent the implication of pancreas, but to restore to the normal the already diseased organ in far as this is at all possible.

DISEASES OF THE LIVER AND BILIARY PASSAGES

JAUNDICE AND HEPATIC INSUFFICIENCY

By O. MINKOWSKI, Cologne

JAUNDICE

GENERAL CONSIDERATIONS

When a patient with jaundice presents himself to our notice, certain peculiarities are conspicuous at the first glance. In a yellow, artificial light the *yellow discoloration of the skin* may easily escape observation, but in broad daylight this is strikingly obvious, and we then perceive the yellow discoloration of the *conjunctival scleræ*; the transudation of the tissues of the lips and the mucous membrane of the mouth with the same yellow coloring matter also becomes visible if we render the tissues bloodless by pressing upon the mucous membrane. This yellow color is readily recognizable upon the hard palate where the greater tension of the mucous membrane makes it pallid.

The yellow discoloration is, in fact, due to an *overflooding of the organism with biliary coloring matter,* as is proven by the composition of the urine which, with its *brownish tint like the color of beer* and the *yellowness of its foam,* shows the presence of bilirubin. The intense staining power of this coloring matter is immediately perceptible on dipping a piece of filter paper into the urine, and is also manifested by the yellow stains which may appear upon the linen of the patient. Certain proof of the presence of biliary coloring is obtained by the positive reaction of Gmelin's test, which is as follows:

We place a specimen of the suspected urine in a test-tube, and with a glass pipette introduce beneath it a layer of concentrated nitric acid to which nitrous acid has been added. The nitric acid should have only a faint yellow color. Chemically pure nitric acid is just as useful as the fuming acid. We obtain an excellent reagent by adding to a larger quantity of pure nitric acid a few drops of the fuming acid, or by heating the pure acid for a short time with a splinter of wood. The continuous oxidation of the biliary coloring matter will then produce at the point of contact of the urine and acid the characteristic *green* zone of biliverdin which, upon allowing the test-tube to stand, is gradually disseminated upward and is subsequently followed by a ring of blue, violet, red and yellow, which slowly rises.

Even with a great dilution of the urine, these bands of color may be distinctly seen if the test-tube is held against a moist piece of filter paper or a plate of ground glass and examined in a clear light.

If we put a drop of nitric acid upon the filter paper through which the urine has been filtered, the characteristic colors will develop in concentric rings. By adding chloroform to some of the urine in a test-tube a yellow color is produced. We may also obtain the Gmelin reaction with chloroform extract by adding commercial nitric acid or bromin water.

The method proposed by Maréchal and Rosin, of covering the urine with a dilute tincture of iodin, will also produce at the point of contact a play of greenish colors which denotes the presence of biliary coloring matter.

Besides biliary coloring matter other constituents of the bile are certainly contained in the urine, chiefly *bile acids*. Complicated chemical methods are necessary to isolate these by Pettenkofer's test.

The urinary sediment obtained by centrifugation shows under the microscope *hyaline casts* which, in the case to be described, were stained a distinct yellow. The presence of albumin in this urine could not be demonstrated by ordinary reagents, nor was sugar present.

The overflooding of the organism with biliary coloring matter is betrayed also by the *intense yellow staining of the blood serum*, which may be recognized in a thin layer of the blood in a glass capillary pipette, particularly on comparison with normal blood serum. In the patient under observation the blood was obtained by means of a capillary pipette after pricking the lobe of the ear, and a Gruber-Widal agglutination test was made with serum which was negative in typhoid cultures as well as in those of paratyphoid.

The serum may also be tested by Gmelin's method. With nitric acid, characteristic colored rings will be observed at the point of contact with the precipitated albumin.

In the case under consideration this accumulation of biliary coloring matter in the organism was coincident with the absence of bile from the intestine. The peculiar, whitish gray color and clayish consistency of the feces are often noticed by the patient.

Yet such appearance in normal feces is not proof of the absence of biliary coloring matter or its reduction products, hydrobilirubin and urobilin. On the contrary, it is demonstrable that the grayish white color is due to a large quantity of fats and soaps in the dejecta. Under the microscope we may discern many fat globules and long fatty acid crystals, also a large number of fine needles of sodium, calcium and magnesium soaps. These soaps give to the feces their peculiar *glistening appearance*

If the feces are rendered acid by an addition of acetic acid, and are then shaken up with alcohol and ether, many of these colorless masse may be dissolved, after which the residue shows a dark brown discoloration due to the undigested remains of meat. Neither bilirubin nor urobilin can be demonstrated in the pale yellow ethereal extract, and, in fact, an occlusion of bile from the intestine may be assumed.

The abnormally large amount of fat in the feces is unquestionably the consequence of absence of bile from the intestine, although it is possible that an obstruction to the flow of the pancreatic juice may be simultaneously operative.

The *constipation and flatulence* of which the patients complain must be referred to an inhibition to the flow of bile. Primarily, the normal stimulation of intestinal peristalsis by the bile is lacking.

Whether the absence of the inhibitive effect of bile upon decomposition may be regarded as the cause of the marked production of gas and the somewhat unusual odor of the feces is questionable. Bidder and Schmidt have assumed that the bile exerts an influence upon intestinal decomposition. Nevertheless there can be no doubt that the bile itself may readily decompose, and it certainly exerts no powerful antiseptic effect. Yet we may reasonably suppose that the absence of bile from the intestine favors the development of certain varieties of bacteria, and thus diverts the processes of decomposition into definite tracts. It may be that the alteration in the composition of the intestinal contents from the lack of bile, for instance, the greater proportion of fat, is the sole cause of the abnormal processes of decomposition in the intestine.

The probability that increased decomposition may occasionally result from biliary occlusion is favored by the increased excretion of ethyl sulphuric acid in the urine which has been noted by various authors (Brieger, Biernacki, Eiger, Schmidt, Böhm). But this is apparently not an invariable finding, since other investigators, such as Röhmann, Pott, and v. Noorden, found no marked variation from the normal in the ethyl sulphuric acid contents of the urine in jaundice.

The symptom-complex of jaundice is met with in very unlike but more or less serious affections: In occlusion of the biliary passages from gallstones, in neoplasms of the biliary channels and in the head of the pancreas, in various cases of diffused and circumscribed disease of the liver, especially in certain forms of hepatic cirrhosis. Icteroid discoloration of the skin and mucous membranes of less intensity is observed in other affections, in general circulatory disturbances, in those produced by disease of the heart, in some cases of pneumonia, in general sepsis, and in other infectious diseases. In all of these cases jaundice appears only as an accompanying symptom in contradistinction to the typical symptoms of the disease by which we determine the character of the affection and its probable course.

The circumstances are different in the case under consideration. Here markedly developed icterus dominates the entire clinical picture, and forms the salient feature of the pathologic condition. Here we are dealing *simple essential jaundice,* with *icterus simplex.*

This diagnosis corresponds with what we glean from the history the following case: The patient, a strong man, 26 years of age, and viously healthy, states that about fourteen days ago upon the occasion a festivity he suffered from a stomach disturbance. The symptoms wl appeared within the next few days did not, as on similar occasions, pass away, but continued; a sense of discomfort in the gastric region disagreeable taste, loss of appetite, a tendency to nausea, as well as gen malaise, lassitude, psychical depression, headache, and mild vertigo sisted. At first there was some fever, also slight diarrhea followed constipation, but no colicky pain. After about a week the yellownes the patient became noticeable to those about him. Subsequently he membered that his urine, even for a few days before, was extremely and had stained his linen yellow. Until the present time the patient been able to follow his occupation, but has felt very weak and has em ated decidedly. Solid food, particularly fatty food, has been repugn to him, but he has had increased thirst and a special desire for cool acid drinks.

Further examination revealed no marked change except the jaund a few scratches upon the skin showed that the patient was annoye itching. There was no elevation of the temperature, which was, on contrary, somewhat subnormal, 97°–98.1° F.; the pulse was slow, 64 t There was nothing abnormal in the thoracic organs. The abdomen slightly distended, the hepatic region somewhat sensitive to pressure. liver showed some enlargement just below the border of the ribs, but not particularly hard to the touch. The gall-bladder could be disti palpated below the border of the liver, and apparently was slightly tended. Splenic dulness seemed somewhat increased, but the spleen not be palpated.

We therefore recognized the clinical condition known as "*catarr* jaundice, which in typical cases, as a rule, runs a benign course.

We have refrained from the use of this designation because, i conception, a definite mode of development of the jaundice has assumed, and this has by no means been proven in all cases. The is based on the assumption that the jaundice is caused by a catarrh flammation of the mucous membrane of the gall-ducts, or of the duod at the mouth of the gall-ducts, which *mechanically inhibits the o of bile.* The finding of a plug of colorless mucus at the mouth common bile duct and the difficulty of forcing the contents of the bladder into the intestine by pressure have, since the time of Vi been regarded as the anatomical foundation for this view.

To-day the plug of mucus is no longer considered to be the act struction to the flow of bile. From the evident lack of biliary imb which the mucus reveals, we conclude that, at most, there was an actual impediment to the flow of bile during life. The mechanical obstruction is revealed by the inspissation of the bile itself as well as by the catarrhal swelling of the mucous membrane in the narrow biliary passages. It is true that little of this swelling is to be seen at the autopsy, but the observations of those who have inspected the mucous membranes during life have taught us that swelling, due to marked hyperemia, is not recognizable after death.

Nevertheless, this assumption does not sufficiently explain the development of jaundice in all of the cases which are included under the term catarrhal jaundice; at least, it does not coincide with our ideas of catarrh of the biliary passages, of *cholangitis catarrhalis.* The symptoms of such a cholangitis are often found in cases in which icterus was never present, and are frequently absent when the clinical picture exactly corresponds to essential jaundice.

It is true there are few opportunities for the anatomical investigation of typical cases of this affection, since, as a rule, their course is benign. But their purely clinical investigation, the nature of their appearance, their frequent occurrence under certain conditions, their relation to other forms of jaundice and to diseases of the liver and biliary passages, taken in connection with the results of experimental, physiologic and pathologic investigation, have recently led to other hypotheses which, although by no means clear, are in many respects far in advance of earlier conceptions.

This change of view not only embraces the nosologic position of this special form of jaundice but also explains the processes brought about in these cases by the transference of biliary constituents into the blood and the fluids of the tissues.

ORIGIN

Before discussing further the subject of jaundice, we must express our present opinions in regard to the development of jaundice in general.

At this time we may maintain that all jaundice is of hepatogenous origin, i. e., that general jaundice cannot occur without an implication of the liver. Every case of jaundice, therefore, is due to *absorption of bile which is formed in the liver.*

The old teaching of "*obstructive jaundice,*" the development of jaundice by an inhibited excretion of coloring matter formed outside of the liver and which can be regarded only as an excretion product, has been completely disproven by the investigations of Stern, Naunyn and Minkowski. But the opinion also that the icterus in "*hematogenous*" jaundice is "*anhepatogenous*" is no longer tenable. There can be no doubt that hematoidin (or bilirubin) may be formed from hemoglobin outside the liver, for example, in extravasations of blood. It is also certain that processes which run their course with a massive dissolution of red blood-corpuscles and an increased destruction of hemoglobin may lead to ja dice, but in such cases, as in hemoglobinemia after poisoning with ars ureted hydrogen, toluilendiamin and other poisons, the transformation hemoglobin into biliary coloring matter takes place in the liver (Sta mann, Affanassiew), and its presence there causes the outbreak of jaun (Naunyn and Minkowski). The latest observations of Ioannovics do alter the fact that by hemolysis the spleen plays an especial rôle in production of jaundice, since this organ is actively implicated in the struction of damaged blood-corpuscles. The designation of such forms jaundice as "hematogenous" or "hematohepatogenous" is even less ju fiable, as the material for the production of biliary coloring matter normally furnished by the hemoglobin. If we desire to indicate the spe mode of development of this form of jaundice, the designations "cythe lytic" and "hemolytic" are certainly preferable.

The most likely cause for the absorption of bile in the liver i mechanical impediment to the flow of bile in the biliary passages. Th fore, in all cases of jaundice an attempt should first be made to ascer the nature of this mechanical obstruction. Naturally it is easy to ass a *"stasis icterus"* when either the lumen of one of the large bil passages is occluded or there is external compression of the biliary pass When, however, the impermeability of the excretory passages canno certainly demonstrated, more or less hypothetical explanations of the struction to the flow of bile may be suggested. The fact has been poi out that the secretory pressure of the bile is comparatively low (accor to Heidenhain in the dog about 200 mm., according to Bürker in the r 75 to 80 mm.); therefore even a slight obstruction to the flow of bi sufficient to cause the absorption of biliary constituents by the b Hence it was believed, as has been stated, that the development of si jaundice could be referred to catarrhal swelling of the mucous mem and to the accumulation of mucus at the mouth of the common bile Jaundice in infectious diseases was attributed to complicating inte catarrh and was also regarded as "catarrhal." When jaundice occ in pneumonia it was assumed that, in consequence of a decrease i respiratory excursus of the diaphragm, an important adjunct for the pulsion of bile was absent. Jaundice in cardiac disease was referr a decrease of blood pressure, or to compression of the finer biliary cha by the dilated blood capillaries. In the case of icterus neonatorum condition was thought to be due to the altered circulatory relations birth, to a patulous ductus arantii, and the like. Hepatic cirrhosi at one time attributed to a compression of the finest interlobular channels by constricting connective tissue, at another time to a cat affection of the finest biliary passages or the occlusion of their by granular pigmented masses, by desquamated and swollen epith and the like. Some authors sought for the explanation of the obstr to the flow of bile in the morphologic changes, the swelling and displacement of the hepatic cells, and the "dislocation of the hepatic cell trabeculæ."

"Hemolytic" jaundice from the effect of blood poisoning was first referred by Stadelmann to an inspissation of the bile caused by the increased destruction of blood-corpuscles in the liver, which thus formed coloring matter. Naunyn and Minkowski therefore employed for this form of jaundice the designation "*jaundice from polycholia*," and Stadelmann proposed as a more concise expression, "*pleiochromia*." It did not escape the attention of these authors that the increased formation of coloring matter was not the only cause of the obstruction to the outflow of bile, but, as Stadelmann emphasized, besides an increased secretion of mucus, the occlusion of the biliary capillaries with decomposed epithelia and hepatic cells destroyed by the poison might also impede the outflow of bile, as well as, perhaps, the compression of the finer biliary passages by swollen glandular cells or dilated blood capillaries which had been subjected to toxic influence.

Although a conception of this kind relegated "polycholic" jaundice to the ranks of jaundice due to mechanical obstruction, many authors have regarded "*polycholia*" as a welcome adjunct by which to explain the different varieties of jaundice whose development from biliary stasis could otherwise scarcely be proven. Above all, icterus neonatorum was ascribed to a suddenly increased formation of biliary coloring material brought about in the first few days after birth (Hofmeier, Silbermann, Hayem) by a massive destruction of the red blood-corpuscles. The development of jaundice from polycholia was also assumed by many authors (Rosenstein, Chauffard, Girode, Banti and others) in certain forms of hepatic cirrhosis as well as in the different varieties of "infectious" jaundice. This was based upon the observation that in such cases, notwithstanding the jaundice, there is usually an intense biliary staining of the feces. Grawitz found that in chronic circulatory disturbances free hemoglobin was demonstrable in the blood serum; he therefore believed that jaundice in cardiac disease might be attributed to polycholia. G. Hoppe-Seyler declared icterus in pneumonia after injections of tuberculin and in Graves' disease to be polycholic, because in these conditions he demonstrated a simultaneous increase of urobilin in the urine and in the feces.

In those forms of icterus in which there could be no doubt of a mechanical obstruction to the flow of bile, some authors went so far as to ascribe the jaundice to the polycholia which caused a destruction of red blood-corpuscles. Browicz, to whom we are indebted for very valuable investigations into the histology of the liver, attempted to prove that a polycholia of this kind was the foundation of *all* forms of jaundice; even in occlusion of the common bile duct he believed that only a mediate influence upon the development of jaundice could be ascribed to the mechanical factor. These theories, however, did not prove satisfactory. On the one hand, the pur mechanical explanations were incomplete. In some cases the intensity the jaundice was entirely out of proportion to the demonstrable obstr tion; in many others, no obstruction of this kind could be determined, a finally, in the liver itself signs of biliary stasis were frequently abse When these signs were present there was undoubtedly a mechanical struction to the flow of bile: Dilatation and flooding of the biliary passag the accumulation of bile in the intralobular bile capillaries, and the posit of biliary pigment in the hepatic cells. On the other hand, the m assumption of polycholia or pleiochromia did not sufficiently explain entrance of bile into the blood and into the fluids of the tissues, unl it were assumed that occasionally the liver ceased to excrete bile normal while at the same time bile formation was increased.

Thus it came to pass in the last few years that a new view recei recognition and a new factor entered into the pathogenesis of icterus; an attempt was made to attribute the appearance of biliary constitue in the blood to *special functional disturbances of the hepatic cells.*

I was probably the first definitely to express this thought, for i discussion at the Congress of Internal Medicine in 1892 I made the lowing remarks: “The special function of the hepatic cell, which ena it to introduce certain products into the bile channels, others into blood-vessels or lymph channels—therefore biliary coloring matter and acids into the biliary passages, and sugar and urea into the blood—is ap ently dependent upon the normal nutrition and normal function of cell itself. Disturbances of this function may exist without mecha hindrance to the outflow of bile, and may result in the transmissio biliary constituents to the blood.” I compared the disturbance in f tion of the hepatic cells here described with the disturbance in fun of the renal cells which occurs in albuminuria, and pointed to the a gous appearance of these forms of disturbance in infectious disease intoxications, in lesions of the nervous system, in circulatory disturba and in parenchymatous implication of the organs.

Liebermeister followed a similar train of thought when, a year he described “acathectic” jaundice, which was said to be due either marked change in the liver cells or because in the process of destru they could no longer retain the bile or hinder its diffusion into the and lymph. Later E. Pick voiced the same opinion when he dec most varieties of jaundice to be essentially due to “paracholia,” a dis ance of the flow of bile in a much more general form. For the under consideration I proposed the designation “*parapedesis of bil* differentiating the close analogy with hemorrhage per rhexin an diapedesin, also jaundice “per stasin” and “per parapedesin.”

Absolute proof of this origin of jaundice is extremely difficult tain. But this hypothesis offered not only a plausible interpretati those forms of jaundice in particular for which former explanations were unsatisfactory, but it coincided with many other common pathologic hypotheses. It was unnecessary to combine mysterious "vitalistic" ideas with the theory of such a "special functional disturbance" of the hepatic cells as might lead to the excretion of their products of secretion in abnormal directions. We know that nutritive disturbances and pathologic changes of the cells not only modify the physical and chemical processes which occur in glandular secretion, but the function of the cells as "semi-permeable membranes" may be so altered as to permit the passage of molecules which cannot pass through the normal cell. And particularly in those varieties of jaundice in which this explanation seemed necessary—jaundice in infectious diseases and intoxications, in circulatory conditions and in disturbances of innervation—such a damage to the function of the cell might, doubtless, readily be supposed. Added to this, the histologic researches of Nauwerck and Fraser, Browicz, and Szubinski afforded an anatomical basis for the assumption of an irregular, intracellular exchange of the different products of the hepatic function.

Thus the teaching of parapedesis of bile soon found general acceptance. Some authors went so far as to emphasize the disturbed function of the liver in *all* forms of jaundice, and attached little importance to the mechanical factor in biliary stasis, particularly in the jaundice occurring in cholelithiasis which was formerly regarded as the type of obstructive jaundice.

Conflicting opinions were not lacking. First, D. Gerhardt opposed the view that in the genesis of simple stasis icterus the main point was the abnormal direction of the cell secretion. Subsequently Hans Eppinger attempted to overthrow the entire principle of parapedesis of bile because, by painstaking anatomical investigations, he determined the factor of biliary stasis to be of much greater significance.

Eppinger devised a method of staining by means of which all the processes of the interacinous biliary capillaries were made distinctly visible, and he employed this method not only to determine the normal course of these biliary capillaries between the hepatic cells and their entrance into the liver cells, even in the human liver, but also to test the action of the biliary capillaries in the different forms of jaundice. He observed first that in pure mechanical stasis icterus—after tying the hepatic duct—the accumulation of bile in the biliary passages led to dilatation, tortuosity, lengthening and varicose distention of the intracellular biliary passages until they finally ruptured and their contents submerged the perivascular lymph spaces. Later he found the same dilatation and rupture of the interacinous biliary capillaries in those cases in which jaundice was due to hepatic cirrhosis, poisoning from phosphorus, or from circulatory disturbances. He believed, therefore, that these cases could only be regarded as special forms of mechanical stasis icterus. The obstruction to the outflow of bile was, however, not in the large bile ducts but in their fin branches, which, as in the case of hepatic cirrhosis, either were compres by proliferating connective tissue or, as in the case of toxic and cyanc jaundice, by coagulated masses of bile, by "*biliary thrombi.*"

Investigations similar to those of Eppinger have recently been m by Abramow and Samoilowicz, who, in the main, confirm the form results. These authors also assume, as an absolute prerequisite for development of jaundice, a free flow of bile in the perivascular lyn spaces.

Elsewhere I recently criticized Eppinger's views, and especially emp sized the fact that, in judging such conditions, the interchange of mate occurring in the organism could by no means be gauged by morpholo observations alone. The appearance of the previously mentioned bili thrombi proves that the secretion of the liver is no longer of nor composition, and that abnormal processes of secretion have already ta place in the hepatic cells. We know that in certain maladies, especi in infectious diseases of the liver, the bile may contain albumin. But these cases especially, notwithstanding an existing jaundice, we freque fail to find in the liver any signs of biliary stasis, even upon mir microscopic examination. Deposits of "coagulated bile," of precipit of biliary coloring matter in the intracellular biliary capillaries and wi the hepatic cells themselves, are not found in cases of recent jaundice only when this has existed for considerable time. They are not the c of jaundice, but the consequence of functional disturbance of the he cells leading to jaundice. Biliary thrombi are, at most, to be rega as predisposing factors for the transference of biliary constituents into blood or the lymph, just as the occlusion of the uriniferous tubule urinary casts is regarded merely as a predisposing factor for a functi disturbance of the renal capillaries which usually causes the retentio urinary substances.

For the present, notwithstanding these objections, I believe it ju able to maintain the importance of an abnormal direction of the he secretion in the transference of biliary constituents into the blood, a therefore briefly define the pathogenesis of jaundice as follows:

General jaundice develops only from the absorption of bile u the liver.

The absorption of bile may be due to the following causes:

1. *An obstruction of the biliary passages which inhibits the outfl bile* (*stasis icterus, icterus per stasin*).

2. *A disturbance in the function of the hepatic cells which resu the excretion of biliary constituents in an abnormal direction* (*dif icterus, icterus per parapedesin*).

Naturally no sharp differentiation of these forms of jaundice acc to their mode of development can be made, since both factors may b usually are, combined: Mechanical stasis of bile leads to injury of the hepatic cells which results in a disturbance of their secretory function. And any damage of the hepatic cells may be followed by a mechanical obstruction to the flow of bile, either because the lesion may at the same time affect the mucous membrane of the intestine or of the biliary passages, and hence obstruct the flow of bile, or because from the pathologically altered consistence of the bile its outflow becomes difficult, or because the finer biliary passages are occluded by a swelling of the hepatic cells, by dislocation of the hepatic cell trabeculæ, by connective tissue proliferation, or by biliary thrombi.

In the incipient stages, as a rule, one or the other factor preponderates, and promotes the development of jaundice:

Mechanical obstruction comes into question in the following conditions:

(a) In *occlusion of the lumen of the biliary channels* by *gall-stones* or *foreign bodies* entering from the intestine (fruit kernels, thread worms, distoma, and the like), by *neoplasms* and *cicatricial strictures*;

(b) In *external compression of the biliary channels by tumors* which originate in the stomach, the intestine, the head of the pancreas, the peritoneum, the portal lymph-glands, in the gall-bladder or the liver itself; by *perihepatic bands*, by *floating kidneys*, by *aneurysms of the abdominal aorta*, of the celiac axis, the hepatic artery or the superior mesenteric artery, in rare cases even by *tumors of the uterus and ovaries*, or by the impaction of *fecal masses* in the right flexure of the colon.

Functional disturbances of the hepatic cells promote the development of jaundice:

(a) In *a disturbance of the circulation of the blood* in the liver due to *cardiac disease*, *thrombosis of the portal vein*, etc.;

(b) In disturbances of innervation of the liver, as, for example, in icterus following psychical emotion, *icterus ex emotione*;

(c) From the effect of *poisons* (phosphorus, arseniureted hydrogen, and others) and *toxic bacterial products* introduced with tainted food (*ptomains*) or those formed in the organism in infectious diseases (*toxins*), in pneumonia, septicemia, relapsing fever, bilious typhoid, yellow fever, syphilis, and the like.

SYMPTOMS

We have seen that the most diverse constitutional diseases and the most dissimilar affections of the liver and other organs may lead, in one or the other of the modes here described, or in both simultaneously, to the symptom-complex of jaundice. The importance of the jaundice in the conception of the pathologic condition of the individual case varies, according to whether this symptom-complex is distinctly developed or less so, and whether the symptoms that accompany it—which at one time may be due to the systemic disease, at other times to a change in the liver or to a simultaneous affection of other organs—become more or less prominent in the entire clinical picture. In many cases we regard jaundic only as a *symptom* or a *complication* of another pathologic condition In others, however, **jaundice assumes the importance of a substantiv affection.**

It is not always possible to draw a sharp line of demarcation betwee this substantive form of jaundice and the jaundice which occurs in organ disease of the liver or the infectious processes or intoxications which lea to jaundice.

The conditions are nowise different from those of other patholog changes produced by the effect of various deleterious agents in the orga ism. Whether, for example, in the individual case, we speak of sept nephritis, myocarditis, and the like, or of sepsis with albuminuria a cardiac insufficiency, depends only upon the prominent implication of in vidual parts of the organism in comparison with the general symptoms.

From this point of view we can readily understand that the numer cases of simple jaundice, no matter how characteristic the mode of th appearance and their development, can by no means always be regar as of the same nature. And, on the other hand, it immediately becon obvious that in simple jaundice, from the benign to the most severe for those with decided constitutional affection and very marked changes the liver, icterus gravis and icterus infectiosus, there is every possi transitional stage. In one instance various factors may act conjoi upon the liver and the biliary passages so that the symptoms of sim jaundice are produced. In another, the same cause may generate eff of varying intensity so that at one time serious, at other times m affections result.

The milder cases of simple jaundice, as already stated, are inclu under the conception of "*catarrhal*" *jaundice.* In fact, *catarrhal cho gitis* appears to be the primary and principal anatomical foundation of pathologic picture which has been described. By an inflammation of mucous membrane of the biliary channels and an increased excretio mucus this cholangitis may obstruct the flow of bile, and thus mechanic lead to stasis icterus.[1]

Cholangitis, like all inflammations of the mucous membrane, ma produced by *toxic* or *infectious* processes of any kind. Deleterious prod may enter the bile *from the blood*, may pass through the liver into biliary passages, or enter into the biliary channels from the intestine. the case first mentioned, *secondary infection* may play a great rôle by entrance of bacteria from the intestine.

But, as has been stated, the theory that jaundice originates from mechanical effects of a catarrh of the mucous membrane has shown itself to be unsatisfactory. Here, too, we must allude to the possibility that cholangitis may be the cause as well as the consequence of biliary stasis.

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[1] In a case of catarrhal jaundice, Kimura estimated the viscosity of the bi 58.24 in comparison with a normal viscosity of 13.21.

It is true we know that bile is sometimes neither aseptic nor sterile, but, under normal circumstances, it certainly contains but few bacteria and these are non-pathogenic. The narrow mouth of the common bile duct, which is frequently washed by the flow of bile, prevents the ingress and development of microörganisms from the intestine.

As soon as the outflow of bile is impeded, the bile duct may be invaded from the intestine by the most varied pathogenic microbes: The bacterium coli, the different varieties of proteus, staphylococci, streptococci, pneumococci, typhoid bacilli and others, penetrate into the bile channels and there find in the " residual bile " a suitable soil for their development. *Thus any check to the outflow of bile may lead to infection of the biliary passages and inflammatory changes in its mucous membrane.*

Thus all mechanical obstructions of the biliary passages, whether occlusion of the lumen of the bile ducts or their compression from without, favor the production of cholangitis. But diseases of the liver which lead to a disturbance of the nutrition and function of the hepatic cells may also obstruct the flow of bile, and facilitate the development of cholangitis. As a matter of fact we frequently find *cholangitis to be a complication of the most varied acute and chronic forms of disease of the liver and bile channels.*

This complicating cholangitis increases the hindrance to the propulsion of bile and promotes the development of jaundice in the diseases we have enumerated, hence jaundice in gall-stones, in hepatic cirrhosis, and the like, has usually been regarded as " inflammatory," and has been attributed to a complication with cholangitis.

Of late, it has frequently been pointed out that these noxious effects, toxic and infectious influences, which we consider the primal cause of cholangitis, not only affect the mucous membrane of the intestine and the bile channels but also the parenchyma of the liver, and, therefore, even *in simple jaundice, an affection of the hepatic parenchyma and disturbance of the hepatic cell function* have been held responsible for the appearance of the clinical symptoms.

Some authors, among them Chauffard, have maintained that the action of *poisons* which are introduced with the food or which develop within the digestive tract by abnormal fermentation and decomposition, is conveyed to the liver by the portal vein. Others, such as Gilbert and Girode, Charrin and Roger, attach the greatest significance to the *local infection* of the liver by the ingress of bacteria from the intestine. Still others, such as Botkin, Kelsch, and Heitler, in these cases believe only in a *general* infection which, by its special localization in the liver or by the action of the toxins which the organism develops in the liver, brings about an obstruction to the flow of bile.

This last conception found a firm support in the observation of cases of jaundice, which are on the increase, in which the action of the infectious agent betrayed itself simultaneously in the *clinical course of the individual case* and in the *epidemic appearance of kindred affections.*

Simple jaundice, as we may perceive from the history previously quoted, is not rarely accompanied by more or less well developed *symptoms of a general infection*—fever, enlargement of the spleen, albuminuria. In these cases we speak of *"infectious jaundice."* In the mildest cases these symptoms of general infection are scarcely perceptible or only temporarily so; often they appear only in the first days of the disease, and are readily overlooked. In some cases, however, they become much more noticeable as the disease progresses, and, finally, there are cases which run their course with the most severe symptoms of a general febrile affection, cases in which jaundice is merely a concomitant phenomenon of the entire pathologic condition.

Such types, the severe as well as the mild, have occasionally appeared in epidemics. This *"epidemic jaundice"* has frequently been regarded as a *special specific infectious disease,* and many authors have attempted to explain *isolated cases of simple jaundice occurring at the same time as "sporadic cases" of this infectious disease.*

Numerous observations of such epidemics, appearing under most varying circumstances, have been reported. These observations, however, by no means confirm the view that the disease is invariably due to a definite *specific* pathogenic agent. It is true that for the development of the different cases occurring in a single epidemic one and the same pathogenic agent might be assumed to be the cause, as, for instance, in those epidemics which Lürmann observed in Bremen occurring after re-vaccination, or those which Rizet observed in soldiers engaged in cleaning the moat of a fortress, etc. But it is by no means proven, or even likely, that in these epidemics the same pathogenic agent was always operative. Not only have the bacteriologic investigations led to a different conclusion, but all which has been learned regarding the cause, the manner of distribution, the incubation of the disease in the various epidemics, is opposed to the view that the same specific pathogenic agent was the cause.

Even the attempt to differentiate from the numerous cases of infectious jaundice a definite group characterized by a peculiar course of the disease as an especial infectious disease, the so-called "**Weil's disease**," has so far been unsuccessful.[1]

In the year 1886 Weil described the first four cases of a peculiar disease running its course "with splenic tumor, jaundice and nephritis," which was characterized by a special febrile course with a tendency to relapses; this affection was subsequently named after him. Since that time numerous publications have appeared concerning the disease; in 1901 Fränkel collected from literature no less than 150 cases. Nevertheless, even to-day, the views regarding the nature of this symptom-complex are wide asunder. Some authors, such as Fiedler, Neumann, and Münzer, share Weil's opinion that it is a special infectious disease due to a specific pathogenic agent. Many others, such as Brosch, Cramer, Stirl, Pfuhl, Fränkel, Nauwerck, Karlinsky, and Soupault, assume that the same pathologic picture is produced by various causes, and that the cases described as Weil's disease have masked various infectious diseases and intoxications: Enteric fever and relapsing fever, infectious intestinal catarrh, tubercular pericarditis, santonin and ptomain poisoning and the like, and many others which have run their course with phenomena resembling Weil's symptom-complex.

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[1] See volume "Infectious Diseases," page 808.

During an epidemic of infectious jaundice in the year 1892 Jäger isolated a special microörganism, the pleomorphic *bacillus proteus fluorescens*, which he regarded as the pathogenic agent. The specificity of this bacillus was doubted. Yet it appears to be a fact that this variety of proteus is capable of producing jaundice, although it has as yet by no means been proven that a definite characteristic course of the disease may be attributed to the action of this special bacillus.

While it is perhaps true that many isolated cases of simple jaundice may be referred to the same origin as cases of epidemic jaundice—nevertheless, the former need not be regarded as sporadic cases of a well marked infectious disease, but, on the contrary, even in these a very different etiology may be assigned for the jaundice.[1]

Naturally these deleterious agents frequently enter the stomach and intestinal canal with the food, and develop within the digestive tract under the influence of abnormal processes of decomposition which the ingesta there undergo. This enables us to understand why, as a rule, the symptoms of gastric and intestinal catarrh introduce the clinical picture, and why the development of jaundice is usually referred to the same etiologic factors which we are accustomed to regard as the causes of gastric and intestinal catarrhs: *The immoderate consumption of food, the ingestion of food difficult to digest or that is tainted, of food too cold or too hot, the abuse of alcohol, refrigeration, infections and intoxications.*

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[1] From this point of view the recent contradictory reports regarding the *agglutinating properties of the blood serum of patients with jaundice* may possibly be understood. Among the cases of jaundice many are due to an infection with typhoid bacilli or other closely related varieties of bacteria.

ACUTE YELLOW ATROPHY

The clinical picture of icterus gravis is, as a rule, associated anatom cally with an *acute diffuse degeneration of the parenchyma of the liv* which leads to the destruction of the hepatic cells and a rapid decrea in the size of the organ which usually is designated as "*acute yellow atr phy of the liver.*" This designation, however, applies only to the appea ance of the liver in the advanced stage of anatomical change which cas running a typical clinical course usually attain. But the intensity of tl clinical symptoms by no means invariably corresponds with the intensity the anatomical changes. Often the patient perishes before the anatomic lesion of this characteristic stage is reached, therefore the term "icter gravis" does not aptly describe the condition known as "acute yell atrophy of the liver."

In these cases of fatal jaundice it is difficult to differentiate the d turbances which are due to the absence of hepatic function and those d to the consequent alteration in the composition of the blood, the *hepa toxemia* or hepatic autointoxication from the toxic effects of biliary c stituents which are circulating in the organism, the *cholemia* from th symptoms produced by the primary *general infection* or *general into cation.*

Until very recently attempts have been constantly made to prove experimental investigation that these severe symptoms depend mainly u the action of biliary constituents circulating in the blood, especially of *bile acid salts.* A few years ago Bickel demonstrated experimentally t the biliary salts when brought directly into contact with the surfaces the cerebral hemispheres prove themselves to be especially potent pois to the nervous system.

Nevertheless it is questionable whether the biliary salts ever attair the organism such a concentration as is necessary in the experimen produce a toxic action.

With some degree of probability, we may refer to cholemia in a stricted sense, that is, to the *over-flooding of the organism with bil constituents,* the following:

The *slowing of the pulse* so frequently observed in jaundice, whic partially due to the direct action of the bile acids upon the heart mu partially to central stimulation of the inhibitive fibers contained in pneumogastric nerve; also certain *milder disturbances* in the *nervous muscular apparatus* which are expressed by itching of the skin, disturb of sight, particularly xanthopsia, headache, general lassitude, musc weakness, and psychical depression; furthermore, a *damage to the kid* which is chiefly revealed anatomically by certain alterations in the epitl and in the convoluted uriniferous tubules, and is clinically manifeste the appearance of albuminuria and the excretion of casts which, as N nagel has shown, may also be found in the urine of jaundice when albumin cannot be demonstrated by the ordinary tests.

Whether the property of the bile acids *to dissolve red blood-corpuscles* is also called into action in jaundice is very questionable on account of the slight concentration of the biliary salts in the organism. Nor is it certain that we can refer to the action of the biliary acids the changes in the walls of the vessels which are the foundation of the *hemorrhagic diathesis,* and which occur in severe cases of jaundice.

On the contrary, in cases of icterus gravis, probably all of the other symptoms of severe autointoxication are chiefly due to a *damage of the hepatic function* and an alteration in the composition of the fluids produced by hepatic insufficiency. For these phenomena are also observed after experimental exclusion of the hepatic function as well as in severe hepatic diseases in which there is no marked jaundice.

The special cause of the toxic effect here produced cannot at present be decided with certainty. Probably, however, the substances whose accumulation in the organism brings about this autointoxication are to be sought for in certain *prior nitrogenous stages of urea* the further development of which occurs in the liver; or in the *toxic products of intestinal decomposition* which, under normal circumstances, are deprived of their deleterious power by passing through the liver; or in the *products of decomposition of the hepatic tissue* itself, which may be destroyed by the action of the same deleterious agent which generates the disease.

SYMPTOMS

If, from the views here expressed, we consider that the difference between benign simple jaundice and that running a fatal course, icterus gravis, is merely a difference in the degree of *intensity* of the action of the same or similar factors upon the same tissue elements, it is immediately obvious that no sharp line can be drawn between these forms of jaundice.

As a rule, these forms are ushered in with the same symptoms—those which we recognized in the patient whose history has been related. Only the subsequent course of the disease varies.

In ordinary cases of *simple jaundice,* the symptoms, as we have seen, last for a short time, generally from two to three weeks, with but slight variation in their intensity, after which recovery gradually sets in. The appetite returns, the patient is more comfortable, the digestive disturbances entirely disappear. The feces begin to show biliary staining, the return of bile to the intestine often being indicated previously by the presence of urobilin in the alcoholic ethereal extract of feces which have been rendered acid. The biliary coloring matter in the urine gradually decreases; often a more decided urobilinuria may transitorily appear. The yellowish discoloration of the skin and conjunctivæ may last longer than the o symptoms. Finally, this also completely disappears, and in three or f weeks after the onset of the disease the patient entirely recovers.

But there may be deviations from this course. Not rarely, particul in epidemic jaundice, digestive disturbances and constitutional sympt are very slight from the onset, or may be absent entirely. Only the icte discoloration of the skin and the urine and the clay-colored stools bet the inhibition to the flow of bile. In such cases the duration of the dis is less, being only from ten to twelve days.

In other and much rarer cases, the disease is very protracted, o lasting three or four months, or even longer. Occasionally, in spite of prolonged course, the reaction upon the constitutional condition is v slight. As a rule, however, these cases gradually increase in severity, produce a decided loss in strength, emaciation, and anemia. Convalesce in such cases may be correspondingly protracted. Finally, however, c plete recovery occurs. Rosenstein reports a case in which catarrhal ja dice lasted more than a year, but terminated in restoration to health. such instances, however, the diagnosis must be considered very q tionable.

Occasionally these cases become more grave. More or less sudde severe symptoms of cholemic intoxication and hepatic insufficiency app combined with a failure in renal and cardiac activity, or the pati perish from the addition of the hemorrhagic diathesis. These cases, th fore, are examples of a transitional change into the critical state *icterus gravis*.

Typical cases of the latter form usually present the picture of ben jaundice only during the first days. From the onset the constitutic symptoms are somewhat more intense, and often enlargement and mar sensitiveness of the liver upon pressure are early noticeable. Splenic tu and albuminuria may be demonstrated even in the first days of the ease. As a rule, however, these cases at the onset do not differ gre from those previously described.

But soon, after a few days, toward the end of the first or during second week, rarely after a longer course, a change ensues: The heada increases and sometimes becomes exceedingly severe, the patient is restl excited, unable to sleep and begins to be delirious. The delirium creases and occasionally assumes a furibund character. The pat screams and tosses about and can hardly be kept in bed. In other c the delirium may be of a more quiet nature, or from the onset ther a tendency to apathy and somnolence. Finally the sensorium is more more benumbed, the patient becomes soporous, and then sinks into deepest coma. During this time there are convulsions in individual mu groups, with grinding of the teeth, attacks of trismus, often even gen convulsions. Vomiting repeatedly occurs, the vomitus sometimes cont d in greater or less amounts. Profuse gastric and intestinal hem- may also appear, and there may be bleeding from other parts ody in addition: Petechiæ and ecchymoses upon the skin and nembranes, severe epistaxis, bleeding from the gums, hemorrhages urinary passages, and, exceptionally, also pulmonary hemorrhage. time, conspicuous changes take place in the liver. The organ s at first enlarged begins to decrease in size. The extent of this may be recognized by examinations repeated from day to day. he organ is scarcely, if at all, recognizable upon percussion. The n the other hand, is usually enlarged.

xcretion of urine is diminished. The urine shows jaundice, con- umin and casts, but little urea; on the other hand leucin and re excreted as sediments, or in testing the urine are readily ated.

g this time the temperature is frequently subnormal; sometimes, it may be normal or even show febrile ranges. The pulse, which appearance of jaundice frequently becomes slow, subsequently is id, smaller, and weaker. Finally, the pulse rate shows extraor- pidity but loss in strength, and it rapidly declines, the respiration irregular and interrupted, pulmonary edema appears, and the erishes in profound coma.

ntire course may be so extremely fulminant that even four or after the appearance of jaundice, and one to three days after e cerebral symptoms have become prominent, death may occur. rare cases a course even more rapid has been observed. A some- tracted course is less rare: The initial stage may last for two or ks or even longer, and after the appearance of the severe symp- duration of life is sometimes from twelve to fifteen days.

e cases with characteristic symptoms which have just been de- favorable course is extremely rare; but several undoubted in- f this kind have been reported. Recovery is then very slow, and nce is exceedingly protracted. Sometimes recovery sets in with n appearance of profuse diuresis ("crise urinaire polyurique,") or a profuse diarrhea. Harmful products are thus apparently from the body. Gradually the hemorrhage ceases, the mind e general condition improves. As a rule, the liver regains its ze very slowly, but occasionally this may be with extraordinary Many months may pass before the debilitated and extremely patients completely recover their strength.

the mildest cases of jaundice, the symptom-complex of icterus y occur secondarily in the course of various chronic diseases of as, for instance, in the different forms of chronic diffuse hepa- e rarely in congestion of the liver, syphilis of the liver, hepatic , and others. In such cases the affection often shows the char-

acteristics of a terminal complication. The disturbances on the pa
the nervous system rarely appear suddenly, but, as a rule, insidiousl
gradually, and they soon increase to the deepest coma or to deliriun
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ETIOLOGY

The *causes* of the disease will decide the character of the path
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temporary complication. In the first group *yellow fever*, above all, must be included, for in this the implication of the liver plays such a rôle that Liebermeister remarked: "Yellow fever is merely an acute parenchymaous degeneration of the liver due to a specific infection, and, aside from he effect of the toxins of the disease upon the liver, there is no reason assume a direct action of the same in other organs." Comparatively ten mild and even severe forms of jaundice occur in *relapsing fever* bilious typhoid"), also in *septic* and *pyemic affections*, but less fre- ently in *enteric fever, cholera, diphtheria, malaria,* and *tuberculosis*.

The occurrence of milder forms of jaundice as well as highly developed ses of acute yellow atrophy in *syphilis* requires especial mention. Where is occurs in the early period of the disease during the time of the erup- on of the secondary exanthems, it occasionally appears in a very charac- ristic manner.

Furthermore, nearly all pathogenic microörganisms may occasionally oduce an outbreak of malignant or benign jaundice, so that we have more reason to regard an "essential" icterus gravis or a "primary" ute yellow atrophy of the liver as a definite specific disease than in the se of simple jaundice.

A predisposition to the severe forms of the disease depends upon the on-resistance of the organism as well as upon the intensity of the effect the poison. This resistance of the organ may have been diminished a previously existing pathologic change. We observe, therefore, as ated, that the mild as well as the severe forms of jaundice frequently cur as complications of hepatic cirrhosis, congestion of the liver, hepatic philis, etc. Yet any impediment to the flow of bile may damage the ver and diminish its power of resistance to poisons and toxins of any ind. *Therefore every jaundice, no matter what its cause, may in its urther course lead to hepatic insufficiency and terminate in icterus gravis.*

As another factor which may cause a special predisposition to jaundice, nd particularly to the severe forms, *pregnancy* must be mentioned. It is emarkable that about one-half of all the cases described in literature f "primary" acute yellow atrophy of the liver occurring in women have een in pregnant women. We cannot regard these cases as solely due to he influence of puerperal septicemia since only a few occurred in the uerperium, the majority having been observed between the fourth and eventh months of pregnancy.

The special predisposition of the pregnant woman to severe attacks f jaundice is revealed by the fact that, in different epidemics of jaundice, ven in those which were largely of benign character, among those who erished with the symptoms of icterus gravis the number of pregnant men was conspicuously large. The French authors (Saint-Vel, Carpen- r), in particular, have described such epidemics.

The cause of this extreme susceptibility of the pregnant cannot be found in the mechanical effect of the gravid uterus, as Klebs has supposed, since the disease occurs even during the earliest months of pregnancy. It is probably the consequence of the changes which the hepatic cells as well as the renal epithelium and other tissue elements undergo during the course of pregnancy.

Brauer, who recently reported a peculiar case in which jaundice recurred during four successive pregnancies, refers the damage of the liver to the preceding action of placental toxins (Veit's syncytiolysins), connecting them with the kidney of pregnancy, with eclampsia, osteomalacia, and other expressions of gravid toxicoses. Brauer attempts to explain *menstrual* jaundice also in a similar manner.

That a *congenital,* often *hereditary, predisposition* may bring about a lessened resistance of the liver and a tendency to mild or severe forms of jaundice is extremely likely. Such a hereditary predisposition to chronic icterus occurring in families is shown in a remarkable manner by certain cases to which I shall briefly refer:

Cases of chronic jaundice appearing in several members of the same family have been described by earlier authors (Murchison). Recent publications are also at hand (Minkowski, Gilbert et Lerebouillet, A. Pick and others). Under the name of "hereditary jaundice" or of "cholémie familiale" very different conditions have been portrayed, and the French authors especially, with their tendency to "diatheses," have attached great significance to the most heterogenous phenomena as expressing hereditary predisposition depending upon family cholemia. In some cases the cause of hereditary jaundice has been sought in a congenital and hereditary constriction of the biliary passages. Pick assumes for his cases either a congenital communication between the lymph channels and biliary passages or a congenital insufficiency of the hepatic cells.

To me it appears questionable whether in all cases of hereditary jaundice a primary change in the liver or in the biliary passages must be assumed. I do not believe this to have occurred in the cases I described as a "peculiar hereditary affection running its course under the picture of chronic jaundice with urobilinuria, splenomegalia and renal siderosis" (*Verhandlungen des XVIII. Congresses für innere Medicin,* 1900).

This symptom-complex occurred in at least eight members of a family in three generations without producing any serious disturbance of the constitutional condition. In one case, upon which an autopsy was held, death being due to other cause, noteworthy structural changes could not be determined either in the liver or in the biliary channels; however, in addition to the marked hyperplasia of the spleen, a very conspicuous deposit of iron was found in the kidneys, which, so far as I am aware, has not been observed in any other condition. At that time, therefore, I expressed the belief that this was due to an especial, transmissible, hereditary anomaly in the metabolism of blood pigment which only seconda-l to disturbance of the function of the liver. During the discussion, r reported similar observations and, in the main, agreed with me.

their clinical course the cases resembled the clinical picture de- by Hayem and Lévy as "ictère chronique infectieux splénoméga- which Gilbert and Lereboullet have also designated as "cholémie le." But the mere fact of the hereditary appearance in successive ions is against Hayem's conception of jaundice as an infectious a conception which was also opposed by Bettmann who described r case.

mann determined periodically in his case a hemoglobinemia without binuria, and therefore considers a congenital abnormality of the be the basis of the disease.

ntly I met another family which shows characteristically the affec- have described. One member of this family, then a student of e, was examined by me twelve years ago. On account of jaundice, uria, and an enormous splenic tumor, I diagnosticated hepatic s, nothing militating against such diagnosis except the absence of logic factor, and, since that time, the continuously undisturbed condition. After the appearance of my publication this gentleman, s since become a physician, brought to me his brother, who since od has also shown icteroid discoloration of the skin, has voided a ine rich in urobilin, and has also had a large splenic tumor. The of the patient is said to have died at an advanced age of hepatic s. The grandmother, four brothers and sisters, and two children the same anomalies from youth; therefore eight persons in four ions. The cases resemble perfectly the first ones I described. They be reported more in detail by Dr. Haal, who has instituted far- g investigations concerning them.[1]

PATHOLOGY

would digress too far were we to discuss more in detail the ques- hich arise concerning the subject under discussion. I shall, there- t enter upon the *pathologico-anatomical findings* in jaundice, but ly remark that the changes which develop in consequence of dis- es of the flow of bile in the liver are only in part to be referred *mechanical and chemical effects of the retained bile.* To a great they depend upon *secondary infection* of the biliary passages and e *decreased power of resistance* of the organ owing to the stagnation as opposed to the various deleterious agents capable of producing acteristics of a terminal complication. The disturbances on the part the nervous system rarely appear suddenly, but, as a rule, insidiously gradually, and they soon increase to the deepest coma or to delirium convulsions; hemorrhages are added, characteristic changes in the ur appear, but are less prominent than in the cases of primary disease. Th may be no decrease in the size of the liver; the fever may be slight entirely absent, the temperature is usually subnormal; death results a few days.

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[1] rding to a communication of Dr. Haal, the number of erythrocytes was l, the excretion of uric acid increased, and the amount of iron in the urine increased.—Three additional cases of the same affection have just been by v. Krannhals in the *Deutsches Archiv f. klin. Med.*, Bd. 81.

Icterus gravis sometimes occurs apparently as a primary disease, wi out any previous symptoms denoting disease of the liver, and run characteristic course; only the autopsy then reveals an earlier affect of the liver the course of which has been entirely latent.

ETIOLOGY

The *causes* of the disease will decide the character of the patholo picture, especially the severity of the clinical symptoms in the individ case and, above all, the *intensity of the toxic action or the virulence of pathogenic agent.* Definite noxious agents are characterized by their cial effect upon the liver. Whether they lead only to temporary functio damage of the liver, or to acute, rapidly progressing, destructive proces or to chronic degeneration and inflammation, depends upon their *mod introduction* into the organism, and upon the *duration and intensit* their action.

Thus, for example, *acute phosphorus poisoning* with only slight t effect may lead to mild transitory forms of jaundice. Larger quant of the poison will produce the clinical picture of icterus gravis and dev the characteristic changes of acute yellow atrophy of the liver, unless other effects upon the organism should cause death before the change the liver develop to such an extent. Phosphorus rarely acts so power in man as to cause a chronic diffuse hepatitis, but changes which c spond to hepatic cirrhosis have been experimentally produced in ani by repeated doses of small amounts of phosphorus.

On the other hand, we know that *alcohol,* which, as a rule, has a intense action upon the liver but more frequently implicates this o produces chronic hepatitis. A fatal icterus gravis or acute yellow at of the liver has been only exceptionally observed after acute intoxic with alcohol. After acute alcoholic intoxication followed by gastric intestinal catarrh, a benign icterus has been seen which was looked as "catarrhal," but which, perhaps, was also dependent upon a action of the alcohol upon the liver.

Among infectious diseases there are some which particularly imp the liver so that the jaundice or acute diffuse hepatitis actually fo part of the clinical picture. In other infections it appears to be c temporary complication. In the first group *yellow fever,* above all, must be included, for in this the implication of the liver plays such a rôle that Liebermeister remarked: "Yellow fever is merely an acute parenchymatous degeneration of the liver due to a specific infection, and, aside from the effect of the toxins of the disease upon the liver, there is no reason to assume a direct action of the same in other organs." Comparatively often mild and even severe forms of jaundice occur in *relapsing fever* ("bilious typhoid"), also in *septic* and *pyemic affections,* but less frequently in *enteric fever, cholera, diphtheria, malaria,* and *tuberculosis.*

The occurrence of milder forms of jaundice as well as highly developed cases of acute yellow atrophy in *syphilis* requires especial mention. Where this occurs in the early period of the disease during the time of the eruption of the secondary exanthems, it occasionally appears in a very characteristic manner.

Furthermore, nearly all pathogenic microörganisms may occasionally produce an outbreak of malignant or benign jaundice, so that we have no more reason to regard an "essential" icterus gravis or a "primary" acute yellow atrophy of the liver as a definite specific disease than in the case of simple jaundice.

A predisposition to the severe forms of the disease depends upon the non-resistance of the organism as well as upon the intensity of the effect of the poison. This resistance of the organ may have been diminished by a previously existing pathologic change. We observe, therefore, as stated, that the mild as well as the severe forms of jaundice frequently occur as complications of hepatic cirrhosis, congestion of the liver, hepatic syphilis, etc. Yet any impediment to the flow of bile may damage the liver and diminish its power of resistance to poisons and toxins of any kind. *Therefore every jaundice, no matter what its cause, may in its further course lead to hepatic insufficiency and terminate in icterus gravis.*

As another factor which may cause a special predisposition to jaundice, and particularly to the severe forms, *pregnancy* must be mentioned. It is remarkable that about one-half of all the cases described in literature of "primary" acute yellow atrophy of the liver occurring in women have been in pregnant women. We cannot regard these cases as solely due to the influence of puerperal septicemia since only a few occurred in the puerperium, the majority having been observed between the fourth and seventh months of pregnancy.

The special predisposition of the pregnant woman to severe attacks of jaundice is revealed by the fact that, in different epidemics of jaundice, even in those which were largely of benign character, among those who perished with the symptoms of icterus gravis the number of pregnant women was conspicuously large. The French authors (Saint-Vel, Carpentier), in particular, have described such epidemics.

The cause of this extreme susceptibility of the pregnant cannot be urea in consequence of a deficiency of the hepatic function. According to my investigations, experimentally excluding the liver, as well as according to recent researches (Lang and others) concerning the prevention of amido-formation in the animal body, it appears likely that the splitting off of ammonia from the amido-acids may take place outside the liver, and only the synthetic change of ammonia into urea within the liver. As a consequence of disturbed hepatic function we may therefore assume at most an increased excretion of ammonia with a simultaneous decrease in the excretion of urea. This has actually been determined in some, but by no means in all, cases of acute yellow atrophy of the liver (Minkowski, Münzer, Fränkel, Senator, Soetbeer).

Diagnostically, however, this sign cannot be utilized. The degree of the excretion of urea and the total nitrogen excretion cannot readily be determined in cases of extreme insufficiency of the liver, because, in the first place, the ingestion of food as well as the digestion and absorptio of food is decidedly limited; secondly, because a toxiferous proteid destruction which cannot be calculated is also operative; finally, becaus the excretion of nitrogen, owing to the severe damage to the kidney whic usually occurs simultaneously, does not appear to be uniform. The relativ increase of the excretion of ammonia may, however, be the result of a excessive production of acid in the organism, an *acidosis,* which is actuall a frequent occurrence.

The abnormal acids which in such cases are usually found in the uri probably owe their origin to an increased destruction of tissue element We must include among these the *oxyacids* derived from the amido-aci by splitting off of ammonia, the excretion of which has been determin in the urine in acute yellow atrophy of the liver, also *hydroparacuma* *acid* derived from tyrosin (the homologous *oxymandelic* acid found Schultze and Riess in the urine in acute yellow atrophy). Probably ma other substances belonging to this category are also found.

Lactic acid (oxypropionic acid), which has been detected in the uri in some cases of severe degeneration of the liver, is probably derived fr a corresponding amido-acid, the *alanin* which has also been found in urine in these cases, as well as amidocaprionic acid and leucin. But excretion of this (muscle) lactic acid is, perhaps, connected with a of liver function, for after experimental exclusion of the liver I no remarkably large quantities of lactic acid in the urine.

Whether the *albumoses* and *peptones* which some authors have fo in the urine in acute degeneration of the liver are also to be regarded the results of the abnormal destruction of tissue elements is at least q tionable. On the contrary, the increased excretion of *purin substa* (xanthin bases and uric acid) may undoubtedly be referred to an incre destruction of cellular nuclei. No special diagnostic significance ca present be attached to these varied occurrences.

The differentiation of simple forms of jaundice from jaundice due to gall-stones is of great practical importance. We cannot here enter into the details of the diagnosis of cholelithiasis. But that this condition is a cause of jaundice should be constantly borne in mind if icterus *develops with pain* or if *colicky attacks* of pain have previously been localized in the region of the gall-bladder, if the jaundice *develops very suddenly and soon disappears,* and if *repeated attacks of icterus* occur in the same person.

Above all, we must consider the possibility of incarceration of stone when the jaundice lasts for an *extremely long time.* Although, as has been stated, there are exceptions to this rule, simple jaundice very rarely lasts longer than six weeks. When the course is more protracted, *cholelithiasis,* certain forms of *hepatic cirrhosis,* and, above all, *neoplasms* of the biliary passages come into question.

Marked enlargement of the liver and spleen generally favors diffuse hepatitis.

The diagnosis of simple jaundice in an *elderly person* always demands the careful weighing of all the symptoms. In these cases neoplasms of the biliary passages and the head of the pancreas primarily come into question.

Close investigation of the various pathological conditions and their careful consideration will usually lead us to a correct diagnosis. But the diagnostic difficulties may sometimes be very great. Simple jaundice rarely fails to be recognized; much more frequently this is diagnosticated when instead another affection, especially cholelithiasis, is present.

PROGNOSIS

The prognosis in jaundice depends mainly upon its cause. In simple jaundice it is usually favorable; but, as already stated, every profound jaundice brings with it the danger of a more or less sudden hepatic auto-intoxication.

The greater the development of the typical clinical picture of acute yellow atrophy of the liver, the more serious the prognosis. The fact that this affection of the liver, up to the most recent times, was regarded by many authors as an absolutely fatal disease, and a termination in recovery as a signal proof of diagnostic error, was due to an erroneous idea that the process was of peculiar nature and produced parenchymatous degeneration of the organ which led to atrophy, and the connection of the disease with the milder forms of jaundice and of acute diffuse hepatitis was completely overlooked. If we consider that we are dealing only with different degrees in the intensity of the disease, it will not surprise us occasionally to find a case running a severe course yet terminating in recovery.

The prognosis in the individual case is decided mainly by the *severity of the general symptoms* and the *implication of the central nervous system.*

The effect of alkaline waters, such as those of Carlsbad, Neuenahr, Vichy, etc., has at present only an empiric foundation, and in cholangitis as well as in other affections of the digestive organs it is attributed to the general "anticatarrhal" action of these waters. But the influence of mineral water treatment upon intestinal peristalsis as well as upon the circulation of the blood and the liver is possibly even more important.

The propulsion of the bile may be promoted by any remedies which stimulate intestinal peristalsis. This probably depends upon the production of contractions in the musculature of the biliary passages, and especially of the gall-bladder, by which the bile previously secreted is more rapidly propelled onward. The action of remedies which have been designated as "cholagogues" is probably limited. As a rule, they are laxatives, such as calomel, rhubarb, aloes, jalap, podophyllin, evonynin and the like. It is possible that the action of olive oil and the oleate salts, of which eunatrol (sodium oleate) has lately been praised, is founded upon this. But it is difficult to understand why these substances in particular should be administered in jaundice when, without them, there is already a deficient absorption in the digestive organs of fats, fatty acids, and soaps. The apparent effect of olive oil in the treatment of gall-stones is probably due to the fact that the calcium and magnesium soaps which have been reduced to soft concrements by the oil have been mistaken for gall-stones.

The only cholagogues which actually increase the production of bile are the biliary acid salts. It is obvious that their administration in jaundice is irrational.

The outflow of bile from the liver may be further increased by remedies which strengthen the abdominal press, and thus compress the liver and the biliary passages. The action of the emetics formerly employed, such as apomorphin subcutaneously injected, probably depends upon this fact, but in general they are not advisable. These effects in a milder degree may also be brought about by physical exercises which favorably and simultaneously influence the portal circulation and the liver.

Careful massage of the abdomen may be very beneficial. Gentle massage of the hepatic region with the flat of the hand or by stroking in small circles, also vibration of the right hypochondrium, or the like, is generally preferable to that brought about by apparatus. Recently, however, the various forms of instrumental vibration and massage have been said to render good service. One author particularly advises shock of the entire body, because he himself was cured of a stubborn jaundice by a prolonged railroad journey.

The method, formerly advised by C. Gerhardt, by which the extended gall-bladder was encircled with the fingers and expressed has quite properly been abandoned, since it is of doubtful utility and by no means devoid of danger.

On the other hand, faradization of the abdominal wall, which occasionally is very beneficial in the treatment of jaundice, is still less harmless. It appears to make no difference whether, as Gerhardt originally advised, one electrode is placed in the region of the gall-bladder and the other horizontally opposite and to the right of the vertebral column, or whether the current is extended to other areas of the abdominal wall.

Active gymnastic exercises are said to promote the flow of bile, particularly deep methodical respiration by which a kind of auto-massage of the liver is attained.

Injections of large quantities of water into the colon are very valuable for the purpose under consideration, since they produce an effect by stimulating peristalsis as well as by direct pressure upon the liver and the gall-bladder. According to Krull, injections of cold water (12° to 18° R.) more powerfully stimulate the intestinal mucous membrane. It is, however, frequently desirable to avoid irritation of the intestine; in such cases water of the same temperature as the body is slowly injected into the bowel with the least possible force. By this means the intestine is thoroughly cleansed and there is simultaneously an increased absorption of water which, for various reasons, is very desirable in jaundice.

In order to restore the normal hepatic function, the best method is to assist the curative processes in the diseased organ by decreasing the labor thrown upon the organ as well as by facilitating the circulation of the blood therein.

The labor required of the liver depends mainly upon the quantity and composition of the food. The necessity of sparing the diseased organ therefore primarily necessitates a limitation of the entire food mass. The fulfilment of this purpose, particularly at the beginning of the disease, is in most cases greatly facilitated by existing anorexia and repugnance to food. Often it is much more difficult to prevent the limitation of food from exceeding the boundaries necessary to maintain the body-weight and the activity of the organism.

As to the composition of the food, it must be remembered that the liver is implicated in the digestion of all organic food products, carbohydrates and fat as well as albuminates. As experience teaches us, however, the absence of bile in the intestine results chiefly in a deficient absorption of fats. It must be borne in mind also that the toxic products which cause hepatic autointoxication most likely originate in changes which proteid substances undergo. In jaundice there is almost always an instinctive repugnance to fat and meat. Therefore, in regulating the diet in jaundice, fats must be limited as far as possible, and when practicable these should be given only in a well emulsified form, as in milk; the ingestion of albumin should be kept within moderate limits and not exclusively in the form of meat; and the total amount of food, principally carbohydrates, should be given in an easily digestible form.

A plentiful supply of water in jaundice is desirable for more than one reason; above all we must here consider that the acceleration of the circulation of the blood in the liver and the absorption of water are simultaneously combined with the dilution of all those substances which, in their passage through the liver, are capable of damaging the cells of this organ.

Alcoholic drinks must be excluded, as there can be no doubt of the deleterious effect of alcohol upon the liver. Other irritants such as pungent spices (pepper, mustard, red pepper, and the like) must also be avoided, since experience has demonstrated their unfavorable effect upon the liver, and also because the relations of the portal vein circulation make it appear probable that, after the gastric mucous membrane, the hepatic cells are most readily damaged by the action of these substances.

Moreover, it has lately been shown experimentally by Kobert and his pupils (Carlau, Jürss) that substances contained in mustard, pepper, nutmeg and parsley (oil of mustard, piperin, myristicin, apiol) exert an especially toxic effect upon the liver which somewhat resembles the effect of phosphorus.

In the nutrition of patients with jaundice the following dietary is advisable: Milk, milk soups, broths, fruit soups and flour soups, very littl meat (150 to 200 grams), without fatty sauce and with but little spice wheat bread, stewed fruit, and light starchy food; tender vegetables i moderate quantity and prepared without fat, especially as purée; weak tea abundance of ordinary water, or alkaline and carbonated mineral water and, if desired, fruit syrups. Naturally, in choosing the diet, we mus as far as possible, consider the individual circumstances.

The rapidity of the circulation of the blood through the liver is powe fully influenced by the ingestion of food, for the amount of blood whic flows through the liver depends chiefly upon the quantity of blood whic passes through the roots of the portal vein. Reflex hyperemia of th digestive organs following the administration of food always results i an increased supply of blood to the liver; but while the resorption digestive products caused by the administration of solid food leads to a increased exercise of the functions of the hepatic cells, water causes on an advantageous increase in the rapidity of the circulation and in t nutrition of the liver, without markedly increasing the labor of the orga Herein probably lies the explanation of the favorable results in jaundi from the administration of water, especially of the mineral water cur and for this reason it appears perfectly reasonable that the use of mine water should be restricted to the early morning hours when the stoma is empty.

It is obvious that the various physical curative methods previously scribed, as well as the stimulation of intestinal peristalsis by laxativ will quicken the circulation of the blood in the liver. And here the ch cause of their effect in jaundice is more readily recognizable than in t mechanical stimulation to the flow of bile.

Finally, one of the principal aims of the treatment must be described, namely, the protection of the organism from the injurious consequences of the impediment to the flow of bile. In pursuance of this aim, the following points are, above all others, necessary:

1. To increase the excretion of the accumulated biliary constituents by stimulating the vicarious activity of other glands.
2. To combat the digestive disturbances due to the absence of bile from the intestine.
3. To treat symptomatically the disturbances arising from the presence of biliary constituents in the blood.

To promote the excretion of the biliary constituents circulating in the organism, the activity of the kidneys comes chiefly into consideration. Here also the plentiful intake of water is the remedy by which these harmful substances may most readily be washed from the body. The diuretic action of the salts which are contained in mineral waters is also beneficial for this purpose. In special cases, particularly with threatening autointoxication, more active diuretics, such as caffein, diuretin, etc., are sometimes of use.

In most severe cases of cholemic or hepatic autointoxication, when the condition of the patient will not permit the copious introduction of water either by the mouth or by the bowel, we must attempt by subcutaneous or intravenous infusions of normal salt solution to restore the diuresis which has threatened to cease.

The excretion of biliary constituents by other secretions, for therapeutic purposes, scarcely comes into question. Biliary constituents are only excreted in the urine and in the sweat. It has been supposed that the yellow discoloration of the body linen frequently observed in cases of jaundice was due merely to the absorption of coloring material from the desquamated yellow-stained epithelia of the skin. But Fr. Müller and Leube have obtained the undoubted reaction for bile in sweat produced by injections of pilocarpin. However, the amount of biliary constituents excreted in this manner is very slight. The favorable influence of warm baths is due rather to their effect upon the circulation of the blood and the entire metabolism, and especially upon the excretion of urine, than upon the stimulation of excretion by the skin.

Biliary constituents pass into the other glandular secretions only when there are pathological changes in the secretions or an admixture of inflammatory exudates therein. In serous effusions there may be a very decided admixture of biliary constituents. In cases of jaundice in which larger effusions of this kind are present, it is more often advantageous to relieve the organism of biliary constituents by aspiration and the evacuation of these effusions than to endeavor to bring about their absorption by drugs.

Digestive disturbances due to the lack of bile in the intestine are most actively combated by such regulation of the diet as has been described.

Good service may also be rendered by laxatives, by mineral water treatment, by intestinal irrigation, and, in suitable cases, by gastric lavage. For the disagreeable taste complained of in the latter, and the subjective symptoms on the part of the stomach, hydrochloric acid or carbolic acid may be employed.

In regard to the symptoms, the itching of the skin produced by the flooding of the organism with biliary constituents is one which requires special treatment. Among remedies which have proven effective and which should be enumerated here are the following: Cold ablutions, perhaps with the addition of vinegar, citric acid, soda, aluminum acetate or a one pe cent. solution of carbolic acid; inunctions with a 4 to 5 per cent. carboli acid salve, 10 to 20 per cent. bromocoll salve, or with a 2 to 3 per cent alcoholic menthol solution.

In stubborn cases the internal administration of potassium bromid (grams twice daily) and injections of pilocarpin (0.01–0.02) or of atropi (0.0005–0.001) are recommended. Often we require narcotics (morphi chloral hydrate, and the like). The frequent use of warm baths is ve beneficial.

The symptoms of severe cholemic intoxication indicate such metho of symptomatic treatment as are based on the general laws of the trea ment of poisoning.

CHRONIC INFLAMMATION OF THE LIVER

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Under the name of chronic inflammation of the liver we include all those inflammatory processes in the liver which are distinct from acute inflammation, hepatic abscess. Usually the term "chronic interstitial hepatitis" is also applied to them, and this designation simultaneously indicates that the condition consists chiefly in an inflammation of the connective tissue. Under such a conception, however, difficulties arise. Undoubtedly there is an increase of the connective tissue in every chronic inflammation of the liver, but this is by no means to be regarded as the primary element. On the contrary, eminent authorities—I shall here mention only Weigert and Ackermann—have affirmed that a degeneration of the hepatic cells is the primary, and hypertrophy of the connective tissue only the secondary, element. Although this view unquestionably has much in its favor, and receives abundant support from the fact that a number of poisons which produce chronic inflammation in the human organism—I shall only mention alcohol, phosphorus and arsenic—positively, directly, and primarily attack the liver cells, producing therein contraction, degeneration, fatty infiltration, fatty degeneration, granular cloudiness, and also cause the disappearance of nuclei and coagulation necrosis, etc., nevertheless, in my opinion, schematism would carry us too far if we were to assume that all increase of the connective tissue, which is the characteristic of chronic hepatic inflammation, is only secondary, that is to say, passive, and not also active. I do not believe that this connective tissue increase is produced solely by the primary destruction of the hepatic cells in such a way that by distention, distribution, and proliferation it fills the spaces present. On the contrary, I think we must take the standpoint that many of the etiological factors of chronic interstitial hepatitis act upon the hepatic cells as well as upon the interstitial connective tissue, at one time chiefly upon one, at another time chiefly upon the other, and, in the main, with such effect that the hepatic cells contract under this influence and perish, while the connective tissue, on the contrary, increases, that is, hypertrophies. I desire, therefore, to express my conviction that in many cases of chronic interstitial hepatitis the connective tissue hypertrophy is not merely a passive one, but is also active, and that it is incorrect to exclude by the name "chronic interstitial hepatitis" our conception of primary degenerative parenchymatous conditions. Important proofs of the correctness of this view are at hand. Degeneration of the hepatic cells, as well as connective tissue hypertrophy, is undoubtedly present in advanced hepatic cirrhosis—this is proven by the investigations of Ackermann (1), Hartung (2), and others—but in itself it is not sufficient proof; for why should not the cell degeneration in these advanced stages of disease be a secondary effect produced by the pressure of the hypertrophied connective tissue? The reason must be found in the stage of onset of the process. Is the affection of the cells, or the connective tissue proliferation, primary? In three such cases which he investigated Brieger (3) found both conditions. Orth states in his text-book that new connective tissue formation is often found without a trace of cellular change. A number of other authors express the same opinion. Minute microscopic investigation in chronic interstitial hepatitis shows that connective tissue proliferation varies in different cases. While in one instance the proliferation, in the main, takes place in the interlobular connective tissue encircling one or more hepatic lobules (annular, insular form, that is, monolobular and multilobular cirrhosis), at other times it is more or less distributed within the individual lobules and between the individual hepatic cells (the intralobular, that is, the intracellular form). Connective tissue is also sometimes deficient in cells, fibrous (older foci), sometimes rich in cells (recent foci). The liver cells themselves sometimes show decided degenerative processes, such as infiltration with fat and bile, or the cells are small and contracted; in other cases they are nearly normal, or even enlarged. In almost all processes of the liver, and going hand in hand with new connective tissue formation, net-like canals are found with lumen which are in part to be considered the remains of normal biliary passages, partly as newly formed bile ducts, or their development may be attributed to a transformation of the epithelia of the true hepatic cell trabeculæ which are flattened, so that, according to this last conception, they are not related to the actual biliary passages, nor do they possess special walls (Aufrecht). It is only certain that these canals have a lumen, and that they may be injected by way of the biliary system (Ackermann). The macroscopical appearance of the liver varies in different cases; sometimes the organ is enlarged, sometimes decreased in size; occasionally smooth, at other times nodular; sometimes it is of normal consistence and feels coarse and hard, at other times it is more doughy (fatty degeneration); sometimes the liver is greatly stained by bile, at other times not; sometimes the organ is dark-brown in color and greatly pigmented, at other times whitish and massively infiltrated with fat.

We shall now briefly discuss the different forms of hepatitis, and their mode of origin.

A. THE VARIOUS FORMS OF CHRONIC HEPATIC INFLAMMATION

The various findings mentioned above have led to the differentiation of many individual forms of hepatic cirrhosis, and the greatest schematic development has been attained in France where some authors have elaborated eight, ten, and even fifteen different forms of hepatic cirrhosis. This number will be still further augmented if we take into consideration the etiology of new connective tissue formation and the clinical course of the disease.

Under these circumstances we may maintain comprehensively that new connective tissue develops in the liver from the following causes:

1. Local, chronic foci of inflammation develop around *foreign bodies* (particles of coal dust) which have entered the liver; in the surroundings of *tumors*; around *parasites* (echinococcus); from implication of the liver, from *chronic inflammatory processes in neighboring organs* (stomach, pancreas); from trauma which produces local injury to the liver, and when, in consequence of a primary hepatitis, a secondary connective tissue proliferation of greater or less extent also takes place in the hepatic tissue. To this category also belong the small interstitial proliferations caused by *foci of bacteria* from various *infectious* diseases (tuberculosis, variola, scarlatina, morbilli, diphtheria, etc.).

2. In connection with the abundant and regular use of pungent *spices* (particularly curry) and the abuse of drastics (Cantani) after *gout*. These modes of development must still be regarded as unproven. The same is probably true of

3. The appearance of cirrhosis after *portal vein thrombosis*, to which Botkin and Solowieff (4) have called attention. Solowieff has endeavored to prove Botkin's view experimentally, but these experiments are by no means conclusive. Thrombosis of the portal vein without hepatic cirrhosis is unquestionably observed very frequently in man, and where these occur simultaneously the assumption is obvious that cirrhosis was primary, and thrombosis of the portal vein secondary. Simple occlusion of the portal vein leads to atrophy of the liver.

4. Cirrhosis in connection with *arteriosclerosis* is maintained by Duplaix (5) who, in fact, does not regard hepatic cirrhosis as an independent local disease, but as an affection which accompanies systemic disease. There is so little upon which to base this view that discussion hardly appears necessary. Arteriosclerotic changes may occasionally be found in the hepatic vessels, and may lead to local interstitial proliferation, but true hepatic cirrhosis in consequence of arteriosclerosis may be regarded as unproven.

5. In chronic states of *stasis hyperemia of the liver* in consequence of continuous pressure from the dilated blood-vessels, an atrophy of the

hepatic cells and hypertrophy of the connective tissue, especially about the central portions of the lobules (hepatic vein), which, however, may also penetrate into the atrophied hepatic cell trabeculæ and even include the interlobular spaces (nutmeg liver), so that at last the picture of atrophic hepatic cirrhosis with ascites, etc., develops. To this category probably belong the chronic perihepatitis and pericardial pseudo-cirrhosis lately described by several authors (H. Rumpf (6) and Fr. Pick (7)). We shall later revert to these forms of disease, and describe them somewhat more in detail.

6. The quite frequent coincidence of hepatic cirrhosis and *tuberculosis* is remarkable, particularly tubercular peritonitis. Wagner (8), Moroux (9) and others report numerous instances of such simultaneous occurrence. Wagner's explanation appears to me to be correct, namely, that cirrhosis is the primary condition, and tuberculosis the secondary; nevertheless, there are distinguished authors (Weigert) who hold a different opinion and who maintain that the inverse relation of these diseases to one anothe is the true one. According to these, therefore, tuberculosis is the primar condition, and causes the cirrhosis.

Tuberculosis of the liver, naturally, is frequently observed without simultaneous cirrhosis. The tubercular nodules in the liver frequentl lead to no inflammatory proliferation of the surrounding tissue wort mentioning. In other cases, however, this is very striking, and when great number of tubercular nodules, which naturally must have existe for a long time, are present, the picture of diffuse interstitial proliferatio develops, and presents some similarity to that of hepatic cirrhosis. Co traction of the organ in this case is very rare, probably because the unde lying condition (tuberculosis) early causes death, for tuberculosis of t liver is but a phase of general tuberculosis. We do not deny the existen of such forms, but certainly primary cirrhosis with the subsequent develo ment of tubercles is more frequent. Occasionally we see this combin with more or less decided fatty infiltration of the liver cells. That tub culosis may lead to fatty liver has long been known, and cannot be regard as singular. At one time this, at another time that, of the patholog conditions enumerated preponderates, and thus varying pictures are p sented. We are, however, scarcely justified—and this is evident from t above brief discussion—as the French maintain (Hanot and Gilbert (1 and others), in differentiating this form of cirrhosis as a special one, a then proposing numerous subdivisions—not less than six. This is certai carrying schematism too far.

7. Here we must mention the *malarial liver* which in Germany is qu rare. We therefore quote the following description from French and It ian literature, without permitting ourselves the right of criticism as to correctness of these findings. It is well known that in profound mala infection a large amount of pigment is formed from the destroyed

puscles, and this is deposited in various organs, particularly in the spleen and liver. The pigment enclosed by leukocytes is sometimes found in the hepatic capillaries, producing thrombosis therein, sometimes within the liver cells, and to some extent also in the interstitial tissue. The liver assumes a rusty brown or dirty grayish-brown appearance. The pigment sometimes gives a reaction for iron (melanin), sometimes not. The liver is rich in bile—as in mild jaundice—enlarged, hyperemic, smooth, and painful upon palpation.

In the acute cases proliferation of the interstitial tissue does not occur, as is the case in chronic malaria. In the latter disease marked enlargement of the liver is found, partly in consequence of hyperemia, partly due to an increase of the parenchyma. The liver cells are large, swollen, opaque, with a large nucleus frequently beginning to segment. If the course of the disease is prolonged, from this stage a second develops in which the liver becomes atrophic. The liver cells degenerate, the connective tissue is but little increased, the organ is flaccid and smooth. Sometimes, however, a different condition is found (Kelsch (11) and Kiener) and the liver is more voluminous, is soft, irregular, covered with small nodules, which are whitish, or golden yellow to greenish, of the size of a pea, projecting from the reddish parenchyma. In these nodules the cellular trabeculæ are greatly thickened (to four times their normal size). The individual cells are very large and opaque, and show one or more large nuclei. The normal arrangement of the hepatic lobules is broken up, the peripheral hepatic trabeculæ being centrally flattened by the pressure of the hypertrophied tissue. Sometimes these nodules resemble adenomata. In about one-third of the cases (probably the oldest forms of the disease) we then observe more prominent interstitial proliferations starting from the interlobular spaces, and resembling the process in ordinary hepatic cirrhosis.

These forms and stages of hepatic disease may be combined in one and the same case to a varying extent and thus produce very different pictures. According to Ughetti (12) the malarial liver in the south of Italy differs nowise from ordinary hepatic cirrhosis, and does not present the peculiarities described by Kelsch and Kiener.

8. Similarly as in malaria, hepatic cirrhosis also occurs in *diabetes mellitus*. Some authors have included both forms—this and the preceding —under the term "cirrhoses pigmentaires." Cirrhosis in diabetes—at least in Germany—is a rare disease; this was first described by Hanot and Chauffard (13) as cirrhose pigmentaire hypertrophique. The patient shows marked cutaneous pigmentation—similar to that in Addison's disease— with cachexia and moderate diabetes. The liver is greatly enlarged and indurated, and shows decided pigmentation as well as profuse cirrhotic connective tissue proliferation. The pigment gives a reaction for iron, and shows itself to be closely related to hemoglobin. Hence we may

conclude that in the organism of the patients affected great and long-continued destruction of red blood-corpuscles plays an important rôle. Quincke (14) called attention to the fact that in diabetics there is "siderosis," i. e., a deposit of iron-containing pigment in the different organs. Occasionally this occurs in consequence of hepatic cirrhosis. Upon the basis of Quincke's investigations, however, the view so widely current in French literature, that siderosis of the liver is the cause of cirrhosis, must be rejected. We fail to see why a diabetic should not have hepatic cirrhosis, and, on the other hand, why a patient with hepatic cirrhosis should not contract diabetes. I am unwilling to acknowledge any causal connection between these diseases. In fact, patients with hepatic cirrhosis and a simultaneous diabetes by no means always show the above mentioned pigmentation. Why diabetes leads to hemosiderosis is not quite clear. Perhaps acidosis, the production of oxybutyric acid, leads to the destruction of the red blood-corpuscles in the organism. I believe this a subject well worthy of discussion. That autointoxication occurs in the course of severe diabetes, in consequence of acidosis, that is, the production of oxybutyric acid (coma diabeticum), is, in my opinion, a positive fact. That the circulation of abnormal products of metabolism in the blood must exert a deleterious influence upon the red blood-corpuscles appears to me to be quite obvious. But we would digress too far to follow this thought at the present time. I desire only to mention that in two patients with diabetes and distinct hepatic cirrhosis, whom I have under observation at the present time, the one without acidosis presents no sign of cutaneous pigmentation, while the second, who has been constantly observed for about three years, and who, in spite of a very strict diet, has about 4 per cent. of sugar in the urine, and is passing three liters in twenty-four hours, has severe acidosis and presents the typical picture of diabète bronzé. The former of the patients just mentioned succumbed after a continuous observation of several months, and in him ordinary cirrhosis hepatis was found without any peculiarities. The amount of sugar in the urine, which at first varied between 7 and 8 per cent. (the amount of urine could not be definitely determined, as the patient was careless), then gradually decreased, notwithstanding the fact that no dietetic measures could be carried out with the patient, and during the last two or three months of his life sugar had entirely disappeared. Besides cirrhosis and diabetes there was also decided cystitis, but no nephritis; periodically considerable blood was found in the urine, and the autopsy proved that this originated in the bladder. The urine never showed the iron chlorid reaction. At the autopsy the pancreas was found to be perfectly normal.

The second patient exhibited marked cutaneous discoloration (grayish-brown) in different areas (face, hands, testicles, penis, skin of the abdomen). The pigmentation has increased but little in the last few years. The liver is large, reaching to the umbilicus, is tough, smooth, and reveals a sharp margin; there is no tendency to contraction. The spleen is not enlarged. For about a year and a half the patient has employed alkalies (sodium citrate) continuously, maintains his weight at about 60 kilos, and follows his occupation. There is no jaundice, no ascites, no edema. That the disease, as reported, rapidly leads to cachexia and causes death from ascites and edema, therefore does not appear to be true in all cases. Certainly there has been no apparent change in the condition of my patient for about three years, and the liver invariably shows the same abnormalities.

In diabetes not only the hypertrophic, but also the atrophic, form (Hanot (15) and Schachmann) is occasionally observed.

That there is an undoubted and distinct pigmentation of the skin in these cases, among which the case of my patient above mentioned belongs, and not, as Quincke believes, chiefly a cachexia, I positively maintain from my own experience.

Siderosis with hepatic cirrhosis also occurs in other diseases without diabetes; for instance, in pernicious anemia (Quincke) and phthisis pulmonalis (Marchand).

9. The Italians, for instance Galvagni and Ughetti (16), have described a form of hepatic cirrhosis as "*hepatitis interstitialis flaccida.*" But our experience regarding these observations is at this time so slight that we can do no more than mention the name.

10. The French (Hutinel, Sabourin (17)) have several times described a type of hepatic cirrhosis as "*cirrhose graisseuse,*" and have differentiated it as a distinct form. This is said to be characterized by a marked fatty infiltration of the hepatic cells as well as the distinct proliferation of the interstitial tissue. The liver is sometimes increased in size, at other times small; this form is found in alcoholics as well as in the tubercular. I do not recognize it as a special variety. Any one who frequently examines cases of hepatic cirrhosis knows that the amount of fat in the cells varies within wide limits, and occasionally may be enormous. Here we have probably only the combination of cirrhosis with fatty liver. Which affection is the primary one is of less importance; that alcoholism produces fatty degeneration as well as cirrhosis has long been known, and that fatty liver as well as interstitial proliferation may be observed in tuberculosis has just been stated under paragraph 6.

11. Chronic inflammation of the liver in consequence of the chronic effect of poisons. When *alcohol* is habitually taken by man in large quantities, it not only produces fatty infiltration of the liver but also hepatic cirrhosis. This statement may be regarded as a positive fact. The only question is whether chronic alcoholism produces different forms of interstitial hepatitis or only that form which we designate as Laennec's atrophic hepatic cirrhosis, or also granular atrophy. Later we shall minutely discuss the fact that, chiefly for clinical, and less for anatomical, reasons,

we differentiate two forms of hepatic cirrhosis, namely, (a) the above described atrophic or Laennec's cirrhosis, and (b) hypertrophic hepatic cirrhosis. Some authors, particularly Rosenstein (18), assume that the latter are not attributable to the abuse of alcohol, while most authors agree in regarding alcohol as the *causa peccans.* The question is so important that it must be discussed somewhat more in detail.

Hepatic cirrhosis is most often caused by spirits, and it is especially noteworthy that the quite large number of children mentioned in literature as addicted to alcohol, and who are found with hypertrophic hepatic cirrhosis, are made drunkards chiefly by the deplorable ignorance of their parent and relatives. But beer and wine also cause hepatic cirrhosis, althoug much more rarely.

In France the immoderate use of absinthe is often alleged to be th cause of hepatic cirrhosis. The following is shown by statistics: That i about one-third of all cases, according to Frerichs (12 patients in 36 as well as Bamberger, the immoderate use of alcohol may be regarded posi tively as the cause. Price (19) found among 142 cases of hepatic cirrhosi 108 occurring in men and 34 in women (i. e., about 3 to 1). Abuse o alcohol was positive in 72 cases, probable in 8 or 9, unmentioned in 5 and positively denied in a few. But in these statistical compilations th two forms of the disease have not been separated. Mangelsdorf (20) foun in 49 cases of hypertrophic cirrhosis that 19 might be etiologically referre to alcoholism, in 5 alcohol was suspected, and in 17 it could not be e cluded. In only 5 cases was there no history of alcohol or syphilis, t latter affection being the positive cause in 3 cases, and likely in 2. Wi the widely prevalent use of alcohol by all classes of the population, it extremely difficult to prove, in the individual case, that it has had deleterious effect and has caused the disease. What is regarded as mu by one person another considers very little. One person who takes habi ually a certain quantity of alcohol regards himself as strictly tempera another who drinks the same quantity suffers from pangs of conscien and regards himself as an alcoholic. But the same quantity of alcoh has a baneful but varying effect upon different persons. Here we cann reckon toxicologically by the kilo of weight. One person on a modera daily amount of alcohol is attacked by hepatic cirrhosis; another worshi Gambrinus, Bacchus, and the god of alcoholic spirits, and goes unpunish all his days. But the cases which Mangelsdorf describes, and others whi I have found in literature, are so positive and conclusive that there c be no question as to the influence of alcohol in the development of hyp trophic hepatic cirrhosis. The consumption of from 3 to 6 liters of wi daily is, nevertheless, a large amount, particularly if we refer to its by women. These reports are so conclusive that we can hardly recon with them the view of Rosenstein (20 a and 65) that, in his cases, alco as an etiologic factor could be excluded. We must emphasize that Ros stein's view is of great importance, since he has seen a large number of cases of hypertrophic hepatic cirrhosis, and it cannot be denied that the frequency of the occurrence of these forms varies greatly in different localities. In spite of the immoderate use of spirits and the great prevalence of atrophic hepatic cirrhosis in Berlin, typical forms of hypertrophic hepatic cirrhosis are quite rare. Therefore, we conclude that some unknown factor besides alcohol must be operative to produce hypertrophic hepatic cirrhosis.

How alcohol causes hepatic cirrhosis has not yet been made clear. It is possible that it first produces inflammation in the vessels while it is passing in a concentrated form from the stomach and intestines into the portal vein vessels, and perhaps it has also a directly deleterious influence upon the hepatic cells. We cannot say more than "perhaps." None of the views previously expressed are positive, and each requires proof. It is said that alcohol is not excreted by the hepatic cells, for, according to the researches of Weintraud, even after the administration of large quantities it cannot be detected in the bile.

The results of Weintraud's investigations are diametrically opposed by the recent experimental inquiries of Brauer (*Zeitschr. f. physiolog. Chemie*, XL, H. 3 und 4). This author, after administering large doses of alcohol to dogs, readily demonstrated the presence of alcohol in the bile, besides quite decided amounts of albumin. These findings should be of great significance in proving the correctness of the view above expressed that alcohol enters the liver unchanged, passes into the bile, and directly irritates and damages the hepatic cells. This continuous damage would finally lead to the destruction of the cells and to the development of hepatic cirrhosis. Alcohol is also found in the urine, but less abundantly than in the liver. Besides being affected by cirrhosis, many alcoholics also suffer from nephritis, and these affections may quite properly be referred to the same cause, namely, alcohol. The secretion of the milk glands, however, does not contain alcohol (Klingemann), consequently nurslings, at least, are protected from the consequences of the abuse of alcohol on the part of their mothers and wet-nurses.

Alison's (21) statistics are also interesting; he found in the country among farm hands the proportion of cirrhosis to alcoholism as 1 to 85; in the city, however, with a sedentary mode of life, it was 1 to 25, and with great bodily activity the number of city cases also decreases greatly, i. e., to about 1 to 42. Therefore, we should send our alcoholics to the country and assign them to strenuous corporeal labor if we desire to protect them from hepatic cirrhosis, and this would not even be injurious; but it would prove as difficult to enforce this prophylaxis as to induce them to renounce their alcohol.

In comparison with alcohol as the cause of hepatic cirrhosis other poisons are insignificant.

In man *phosphorus*, more than any other, destroys the hepatic cells and produces marked fatty infiltration. It frequently causes the anatomical picture of acute yellow atrophy, but rarely true hepatic cirrhosis.

Arsenic, antimony and *chloroform* are also said to produce hepatic cirrhosis in man, and, according to Langowoi (22), this is also true of cantharides. Here it seems fitting to consider somewhat minutely the *experimental investigations* which have been made in the hope of solving the many questions under discussion which have just been enumerated. An attempt was made to produce hepatic cirrhosis in animals, and for this purpose various poisons, especially alcohol, phosphorus and chloroform, were used. Unfortunately, as we must admit at the outset, most of these endeavors have been fruitless.

It has been impossible to produce hepatic cirrhosis in animals by administering alcohol. This is shown by the invariable negative trials of Strassmann (23), Afanassiew (24) and v. Kahlden (25). Fatty liver was induced, but not hepatic cirrhosis. In my opinion these results contradict the earlier and positive reports of Dujardin-Beaumetz, Pupier, Strauss and Blocq. Afanassiew claims that he has produced degeneration of the hepatic cells by prolonged and large doses of alcohol, especially in rabbits, while v. Kahlden denies such a pathologic finding.

Perhaps the toxic effect of alcohol with large, individual doses was not long enough continued.

H. Mertens maintains that he has lately produced typical hepatic cirrhosis in rabbits by keeping the animals continuously in an atmosphere permeated with alcohol.

According to Mertens (26), chloroform in subcutaneous injections has a much more powerful effect. He was able to produce typical hepatic cirrhosis, and to determine that the liver constantly became harder, more granular, and more contracted. The liver cells suffered first; they became opaque, atrophic, and showed fatty degeneration, being permeated with vacuoles. Later, starting from the portal vessels, connective tissue developed which became diffused through the hepatic lobules toward the center (the hepatic veins). By the kindness of Professor Heymanns in Ghent, where Mertens worked, I was able to obtain sections of such livers for examination. These livers showed typical granular atrophy with the above described microscopic findings. Nevertheless, I am very skeptical in regard to experimental investigations in rabbits on account of my own extensive experience. These animals frequently have diseased livers, and consequently we are easily led astray in regard to, at least, the early stages of cirrhosis.

I do not altogether countenance the investigations of Boix (27) who, by the introduction of small amounts of butyric acid (for three months), acetic acid (for thirty-five days), and also of lactic acid and valerianic acid, produced typical atrophic hepatic cirrhosis in rabbits.

Recent experiments have cast a doubt upon the apparently positive production of phosphoric hepatitis observed by Wegner. Krönig (28), as well as Ziegler and Obolensky (29), found almost invariably marked epithelial degeneration, but the inflammatory phenomena were only moderate, and, according to my investigations, the conditions resembled those produced by toluylendiamin. Ziegler and Obolensky expressly state in their report: "It appears impossible, from the investigations at hand, to draw conclusions from poisoning by phosphorus and arsenic as to the genesis of hepatic cirrhosis in man. At present the latter can only be determined by the histologic investigation of cirrhotic livers."

Aufrecht (30), it is true, holds a different opinion. He maintains that he has seen connective tissue hypertrophy follow phosphorus poisoning without any epithelial degeneration. I reiterate the fact that Aufrecht experimented on rabbits, but, as mentioned above, in these animals such experiments are not conclusive.

We have still to mention four other forms of chronic inflammation of the liver, which will be explicitly described later:

12. Cirrhosis hepatis, that is, Laennec's cirrhosis, which has several times been mentioned.
13. Hypertrophic hepatic cirrhosis.
14. Biliary hepatic cirrhosis.
15. Syphilitic hepatic cirrhosis.

From a clinical rather than from a pathologico-anatomical standpoint it appears necessary to differentiate the two fundamental forms of hepatic cirrhosis, (a) the atrophic and (b) the hypertrophic hepatic cirrhosis, as two contrasting types, between which are numerous transitional forms; therefore they will be separately considered.

B. CIRRHOSIS HEPATIS—LAENNEC'S CIRRHOSIS—GRANULAR ATROPHY OF THE LIVER

This form of hepatic cirrhosis receives its name, which was first given it by Laennec, from the fact that in advanced stages it leads to contraction which causes the anatomical condition to resemble the granular atrophy of the kidneys. Etiologically this form, as described above, may chiefly be attributed to the abuse of alcohol, hence the affection preponderates among men. Although all clinicians are unanimous on this point, it must, nevertheless, be stated that there are cases of hepatic cirrhosis in which the abuse of alcohol cannot be proven, nor can any of the causes enumerated for the appearance of the affection be found. The question, therefore, arises whether or not, as with alcohol, other toxic products in large quantities (products of metabolism) may not occasionally find their way from the intestine into the portal vein system, and there exert a deleterious influence upon the liver which may result in hepatic cirrhosis. We all know that in the human organism an antitoxic function is quite properly ascribed to the liver, and the inference is obvious that occasionally under

SYMPTOMATOLOGY AND COURSE OF THE DISEASE

The disease certainly exists for a long time—occasionally for years
without giving rise to symptoms, and is often discovered while examini
patients who come to us in consequence of some other affection. In ke
ing with the etiology, symptoms on the part of the gastrointestinal ca
resembling those so frequently present in alcoholics are the first complai
of the patient. These consist of nausea, anorexia, eructations, vomiti
constipation, sometimes even diarrhea, pressure and a sensation of fuln
in the epigastrium, even pains in this region, etc. The enlarged li
causes distress by pressure upon the neighboring organs. The patients
debilitated, the general condition suffers. Pain is rare in true atrop
hepatic cirrhosis. It occurs only when the inflammation especially atta
the capsule of the liver, and this is seldom. In the course of the dise
irregular rises of temperature are occasionally noted, which come and
and for which no cause can be assigned. Severe symptoms are observ
only in the later stages, when disturbances appear which are due to t
portal vein circulation. Mild jaundice may be present, but does not nec
sarily belong to the picture of atrophic hepatic cirrhosis. In conseque
of the destruction of so many small portal vein vessels, there is stasis
the blood in the trunk of the portal vein which reacts upon the stom
and intestine. The consequence is an increase of the catarrhal sympto
in both organs and the appearance of ascites, which then becomes pro
nent in the picture of hepatic cirrhosis and continues permanently. I
tention of the abdomen, and an increasing sense of weight and ful
in the same, are frequently the first symptoms which the patient obser
The ascites may develop to an extraordinary extent, and by forcing up
diaphragm as well as hindering respiration may cause the patient m
distress. Frequently edema of the lower extremities follows. With
constantly increasing symptoms on the part of the gastrointestinal ca
the nourishment is deficient and the patients rapidly lose weight.
enormously distended abdomen forms a striking contrast with the
emaciated almost to a skeleton. Hemorrhages occur in consequenc
decided stasis of blood in the esophagus, stomach, and intestines. T
are occasionally only of the capillary form, so that hemorrhagic feces
the vomiting of black masses are observed. But these hemorrhages
not infrequently be also very copious, and threaten life; then large am
of pure blood are vomited, and the dejecta are of a black, tarry color.
the autopsy ruptured varices are found to be the cause of this, espe
in the lowest portions of the esophagus; but pure parenchymatous he
rhages may also endanger life through their massiveness. The nut
of the walls of the vessels also suffers and leads to hemorrhages in va
organs, such as the skin, the mucous membrane, the retina, etc.;
epistaxis is also sometimes observed. The slight jaundice is

due to compression of the biliary channels in the liver and to biliary stasis due to the resorption of bile, in part it may be referred to duodenal catarrh.

The examination of the abdominal organs with an existing ascites is very difficult, and often is only possible after the fluid has been withdrawn. A hard, contracted, nodular liver is then usually felt; sometimes the organ is so small that it can no longer be palpated.

The condition of the spleen in hepatic cirrhosis is of importance. Frequently it is enlarged to two or three times its normal size; and this is a factor of great significance in the diagnosis of the affection, particularly in its last stages when the liver can no longer be palpated. Enlargement of the spleen is not only due to stasis of the blood in the portal vein, but to direct hyperplasia of its tissue, with an increase of the connective tissue. We must, therefore, assume that active processes are going on in the spleen, similar to those in the liver. Sometimes, however, splenic enlargement is absent, particularly if the capsule is tough and thickened.

Ascites.—Ascites requires a somewhat more detailed description. The fluid fluctuates in the abdominal cavity, and is to be attributed to the passage of fluid through the intestinal serosa into the peritoneal cavity. According to Quincke (32) some of the transudated fluid is poured out by the lymph-vessels of the diaphragm and of the parietal peritoneum, and this is independent of the portal vein circulation. When the ascitic fluid is withdrawn it is usually clear, amber-colored, with a specific gravity of 1.012 to 1.014, and contains from 0.6 per cent. to 1.2 per cent. of albumin. There is scarcely a trace of sugar or biliary coloring matter. Turbidity to any extent points to complicating inflammation of the peritoneum; blood may appear in small or in large quantities in consequence of stasis hyperemia of the serosa.

In addition to the previously mentioned stasis, dilatation of large venous areas which have a collateral relation to the portal vein, or may become thus related, plays an important part in hepatic cirrhosis. Notwithstanding the obstruction to the circulation, large quantities of blood from the portal vein system may be poured into the veins of the body and thus the portal system is greatly relieved. It appears, however, that the compensatory importance of this collateral development is greatly exaggerated; this value, at least clinically, is rarely observed.

Collateral Circulation.—1. The communication of the mesenteric veins with the veins of the abdominal walls. 2. The connection of the gastric veins with the inferior esophageal veins and, through these, with the azygos vein. 3. The conjunction of the internal hemorrhoidal veins and the hypogastric veins. 4. The communication of the veins of Glisson's capsule with the veins of the diaphragm through perihepatitic adhesions. 5. A dilatation of the incompletely obliterated umbilical or paraumbilical veins which run along the ligamentum teres, from which point blood may then be carried by the portal vein to the veins of the abdominal walls, particularly to the epigastric vein. These veins of the abdominal walls are more or less markedly dilated, and form a figure which bears a remote resemblance to *the head of Medusa* (*caput Medusæ*), and accordingly has received this name. The caput Medusæ, at least in its well developed form, is by no means frequent; we must not mistake for it simple dilatation of cutaneous veins, such as the superior and inferior epigastric veins combined, which represent nothing more than collateral tracts of the compressed inferior vena cava; these may be found in any form of abdominal dropsy, and not only when they are the consequence of an implication of the portal vein circulation.

Other and rarer collateral formations, for example, communication between the paraumbilical and the right iliac vein by a vein of the abdominal wall (Drummond), require no further mention.

The *pulse* is small and somewhat accelerated, but it is regular unless there are complications on the part of the heart.

The *respiration* is accelerated when ascites is present because the diaphragm is forced up, and its action is limited.

If death does not take place early from intercurrent diseases and complications, this is preceded by symptoms of increasing weakness and exhaustion. Not rarely the patient falls into a comatose condition with delirium, general convulsions, etc., and from this he never awakens. This gives us the impression that we are dealing with a condition resembling cholemia, such as is observed in long-continued stasis jaundice, or in acute yellow atrophy of the liver. As jaundice is absent or very slight in the affection in question, this cannot be attributed to intoxication with constituents of the bile, but it must be due to other products of metabolism which are not yet sufficiently known. The antitoxic function of the liver is unquestionably not its least important one—this we have previously referred to. But in this respect, as well as in many others, the organ cannot function on account of the constant and increasingly rapid disappearance of its parenchymatous cells. The toxins—probably entering from the intestine—penetrate the protective wall, flood the organism, and produce the condition which we designate as autointoxication. Perhaps the same substances, and not biliary constituents, cause the condition known as "cholemia" or "acholia."

Of the nature of these toxins we have, unfortunately, as yet no accurate knowledge, and in my opinion here, i. e., in the study of this toxin production, scientific investigators should lead the way. Up to the present time, we have considered only ammonia, the aromatic products (phenol, skatol, indol, etc.), the alkaloidal products (putrescin, cadaverin, neuridin, etc., which we have included under the name diamin, and a knowledge of which we mainly owe to the pioneer investigations of Brieger), and the products of metabolism of the bacteria from the intestine which have a toxin-like effect, that is, in other words, the products of proteid decomposition.

In regard to ammonia, the investigations of Nencki, Pawlow, Hahn and others in dogs, with Eck's fistula (excluding the portal vein circulation) should be borne in mind. In their experiments upon animals, these investigators observed symptoms which closely resembled those of cholemia, particularly when nitrogenous food was administered.

Many authors (Heger (33), Schiff (34), Lautenbach (35) and others) found that the liver not only retains certain poisons which are introduced with the blood (nicotin, strychnin, hyoscyamin, etc.) but also destroys them or transforms them into non-toxic substances, or, at least, stores them up.

Special credit for working out and elucidating this question is due to Bouchard (36) and his pupil Roger (37). According to the latter the liver acts upon poisons in three ways: 1. It stores them (metallic poisons such as copper, iron, arsenic, etc.). 2. It excretes them with the bile (a rarer and less active process). 3. It renders the poisons more or less inert, i. e., destroys them or changes them into non-toxic combinations (vegetable alkaloids, peptones, toxic products of intestinal decomposition, ptomains).

This protective action of the liver, according to Roger, is due to the glycogen it contains, with which these poisons combine, and thus become non-toxic. It must be confessed that these teachings have not been generally accepted, and that they cannot as yet be regarded as incontrovertible. We shall see later, when discussing the treatment, particularly the dietetics of diseases of the liver, in how far we may utilize these views. Other experimental investigations to determine the principle on which is based this protective action of the liver toward poisons, I shall not discuss. They have given us no additional information.

Bouchard attempted to prove this important property of the liver indirectly by testing the toxicity of the urine in various diseases of the liver, and comparing it with the normal. He started with the hypothesis that the soluble poisons circulating in the body were excreted in the urine. If the liver had an antitoxic action, this must be limited in hepatic diseases, and, in consequence, the urine must be more toxic (the "urotoxic coefficient" increases). He found his deductions confirmed, and he drew from these many conclusions, some of which, however, are very questionable. If we accept all that has been stated, notwithstanding the criticisms at hand as to the results of experiments, we can no longer doubt the influence of the liver upon numerous poisons (accordingly upon those also which reach it from the intestine).

The investigations of metabolism in hepatic cirrhosis have not been very extensive, but, nevertheless, we have obtained some very remarkable results. In regard to the metabolism of nitrogen in hepatic cirrhosis, Hallervorden (38) has occasionally found very large quantities of monia in the urine (up to 2.5 in the daily quantity). I also (39), following me many other authors (Fawitzky (40), Gumlich (41), v. N den (42), Münzer (43), found the quantity of ammonia increased comparison with the total amount of nitrogen (10 to 18 per cent. inst of the normal 2 to 5 per cent.), but the absolute figures very ra reach the amount determined by Hallervorden (Mörner (44) and Sjöq found in one case 2.4 grams). Yet from this alone we cannot detern a diminution of the urea-forming function of the liver, which, on contrary, appears to be extraordinarily active, usually to the end. besides these large quantities of ammonia, large amounts of urea also formed (30 to 40 grams in twenty-four hours). With the decreased cretion of urea which may be regarded as the consequence of a defic ingestion of food, of inanition, the excretion of ammonia is also lesse The relation of urea to the total amount of nitrogen of the urine is but little altered (77 to 91 per cent. as compared with the normal of to 90 per cent.). Some time ago I (45) expressed the conviction other factors, and particularly an increased excretion of acid, caused increased excretion of ammonia. In the urine I, as well as v. Noor occasionally found sarcolactic acid, as has lately Calabrese (46), w v. Jaksch demonstrated fatty acids, particularly acetic acid, valerianic a butyric acid and propionic acid. Weintraud (47) administered amm up to 9 grams in hepatic cirrhosis, and found no increased excretio ammonia, but only the requisite and expected increased excretion urea. Therefore, if urea is produced in the liver alone and not other organs, which, however, is unlikely, we must not conclude f these findings that this important function is sufficiently performed by remaining parts of the hepatic tissue, and that life ceases before the di leads to marked and distinctly recognizable anomalies in urea forma The recent conclusions of Schittenhelm (*Deutsches Arch. f. klin. Med* LXXVII, H. 5 u. 6) in the main agree with my views; he also r an increase of the excretion of ammonia in the urine in destructive he affections, and refers this to the acidity of the organism. Pepto positively absent from the urine in hepatic cirrhosis; albumoses are sionally found, but they are rare and in insignificant amounts. Uric excretion is within normal limits, as well as the alloxur bodies (prior s of uric acid) which have lately come within the circle of scientific i tigation. The finding of leucin and tyrosin is more than questio von Jaksch (48), who has made very extensive investigations in r to the nitrogen excreted in the urine in hepatic diseases, has come t conclusion that, in the main, it appears as urea, and that other nitrog products are not found in increased amount.

The heat-generating function of the liver must naturally suffer a as the activity of the organ is decreased. As, however, the normal fu of this organ in proportion to the heat-producing power of the entire organism is not definitely known, in pathologic processes, naturally, little can be said on this point. I believe that this function of the most important glandular organ of the body is not insignificant. However, there is as yet no possibility of our determining the effect of absence of its function in the human organism.

Sugar is sometimes found in the urine of these patients. Yet, on account of the rarity of the finding, I do not regard it as in organic relation to the underlying affection (hepatic cirrhosis), but merely as an accidental complication.

Steinhaus (49) found in the majority of cases of hepatic cirrhosis investigated by him (in 11 out of 12) distinct connective tissue proliferation of the pancreas resembling that of the liver, and he believes that occasional diabetes in such patients may be attributed to this condition. These observations, therefore, coincide with the opinion expressed above that diabetes is here not directly due to the hepatic disease.

Alimentary glycosuria has also been observed, but too rarely for us to deduce diagnostic conclusions therefrom, as do the French authors. This is true, at least, regarding grape-sugar (intake and excretion). With the intake of levulose, the condition is different (H. Strauss (50), Lépine (51)), for it has been observed that patients with disease of the liver usually develop levulosuria in contrast with normal persons. In 26 of 29 patients examined by Strauss, this levulosuria could be demonstrated.

As, however, according to the reports of Landsberger (*Deutsche med. Wochenschr.*, 1903, Nr. 32), similar conditions are not infrequent in healthy persons, our anticipation of having found an important aid for the diagnosis of alimentary levulosuria has met with disappointment.

COMPLICATIONS

Alcohol, which is the chief cause of hepatic cirrhosis, also damages a number of other organs, such as the heart, kidneys, vessels, etc. Combined with hepatic cirrhosis we also frequently find myocarditis, nephritis, and arteriosclerosis, but rarely pachymeningitis or chronic meningitis. The other complications of hepatic cirrhosis, such as portal vein thrombosis and fatty liver, have already been mentioned. Myocarditis with its sequels, edema, congestion of the liver, etc., as well as nephritis, is often the cause of the fatal termination of our cases.

The not infrequent combination of hepatic cirrhosis and primary carcinoma of the liver is remarkable, as is the connection between hepatic cirrhosis and peritoneal tuberculosis which has been referred to previously.

DIAGNOSIS AND PROGNOSIS

Easy as the diagnosis of hepatic cirrhosis may be in many cases, in others it may be most obscure. In alcoholics with enlarged hard liver, recognizable enlargement of the spleen, and moderate ascites, the diagnosis presents no special difficulty. This only becomes perplexing when we are dealing with patients in whom marked ascites makes palpation of the abdominal organs impossible. Then we must first determine whether a general circulatory disturbance is the cause of the ascites. If the heart, lungs and kidneys are normal, if there is no general edema, it is exceedingly likely that there is a circulatory disturbance in the course of the portal vein, and, when combined with chronic alcoholism, hepatic cirrhosis may be presumed to be the cause. But only a probable diagnosis can be made in such cases. For portal vein stasis may be due to many other causes (compression of the portal vein by tumor, thrombosis of the portal vein, carcinoma of the peritoneum, of the omentum, etc.). When the ascitic fluid is removed, and we are able accurately to investigate the abdominal organs, we frequently arrive at a correct diagnosis.

The differential diagnosis between hepatic cirrhosis and chronic, as well as tuberculous, peritonitis is especially perplexing. The latter affection combined with hepatic cirrhosis is not so rare as was formerly supposed. The proof of tuberculosis in other organs, the character of the ascites, which in peritonitis is frequently hemorrhagic and also shows a higher specific gravity with a greater amount of albumin, will in many cases lead to a correct conclusion, but we must admit that errors in diagnosis cannot always be avoided. The existence of a chronic perihepatitis, perihepatitis chronica hyperplastica, must also be considered in the differential diagnosis. The great thickening of the capsule leads to compression of the organ, to disturbance of the portal vein circulation, and to destruction of the hepatic parenchyma. The organ, in consequence of the marked contraction of its capsule, becomes small, atrophic, hard, and ascites appears; in brief, a picture which is very difficult to differentiate from atrophic hepatic cirrhosis. A similar affection is described by Pick (*Zeitschr. f. klinische Medicin,* Bd. 29, Heft 5 u. 6), pericardial pseudo-hepatic cirrhosis, i. e., perihepatitis chronica hyperplastica combined with chronic pericarditis and obliteration of the pericardium.

Duration.—The *duration of the disease* cannot be determined with certainty, since, on account of the mild symptoms, the time of onset is not known. In this respect, therefore, individual authors express very varying opinions, and they estimate the duration of life at between one and ten years. The earliest possible recognition of the disease, the whole state of the patient, his circumstances, the avoidance of everything injurious, and, particularly, abstention from alcohol, etc.—all, naturally, have an important bearing upon the course of the disease.

Prognosis.—As to the prognosis in a positive case of atrophic hepatic cirrhosis, all authors are unanimous in declaring the disease to be fatal. Yet many cases of recovery, even in advanced hepatic cirrhosis with ascites, have been reported (I shall only mention Liebermeister, Rosenstein, Semmola, Leichtenstern). Naunyn especially emphasizes that, in his experience, the cure of atrophic hepatic cirrhosis is sometimes spontaneous. Ehret, upon the basis of one of his own cases, has recently expressed himself on this point most explicitly, and entirely agrees with Naunyn. I have never seen an actual cure of atrophic hepatic cirrhosis. Where this appeared to be the case, there was some doubt as to the diagnosis. Syphilitic hepatic cirrhosis will be described separately.

TREATMENT

The treatment is, in the main, symptomatic. The removal of the etiologic cause, the early and entire withdrawal of alcohol, and the forbidding of pungent spices are most important; in malaria we employ quinin, in syphilis potassium iodid, and even mercury given cautiously. Where syphilis cannot positively be determined, potassium iodid should be given a trial. The patients should not be exposed to the deleterious action of lead, arsenic or phosphorus. As soon as the pressure of ascites causes distress, paracentesis abdominis is indicated; early puncture, however, is not advised; the value of this is very problematical. The first deposits of the newly formed ascitic fluid are always most rapidly excreted. When the pressure of fluid in the abdomen has become distinctly positive, accumulation takes place more slowly. By early and, consequently, frequent puncture, a large quantity of albumin is withdrawn from the body, and this hastens the destruction of the patient. In contrast with this is the value of early puncture in decreasing venous stasis in the abdominal organs; this is supposed to hasten the establishment of the collateral circulation between the portal vein and the organs of the body, etc., which is so desirable to the patient, but is as yet by no means proven. In regard to the necessity of tapping, I determine this from the symptoms of the patient, and, since general rules cannot be given, I can only advise it. In some patients we must perform this little operation early and often; in others enormous quantities of fluid in the abdomen occasion no special symptoms.

When the patients complain much of abdominal pressure, and the cardiac activity and respiration are implicated by the displacement of the diaphragm, aspiration must be performed. Not only these difficulties, but many others, then seem to disappear as if by magic. The appetite is stimulated, a sufficiency of food is ingested, the bowels become regular, and diuresis improves. Aspiration is often followed by an amelioration of the entire condition which lasts for some time.

In atrophic nutmeg liver due to diseases of the heart, lungs, and kidneys,

the treatment of the underlying affection, particularly of the heart, m
be our first object. Digitalis is then the most valuable remedy, and
must attempt by diuretics to prevent the accumulation of fluid or to rem
that which has been formed. Cream of tartar, potassium acetate (alw
in large doses, 10, 15 to 20 grams a day), juniper, and special diure
such as boneset tea (50 grams macerated, placed in a liter of cold w
and allowed to boil for three or four hours, then poured off and filte
always to be freshly made, and the entire amount taken in twenty-f
hours). Urea, which was recently strongly advised as a diuretic by Kl
perer (10 to 20 grams a day), I have not found specially valuable.
my experience it acts no better than the remedies mentioned above w
given in the same quantity, and these may be prescribed when the kid
are normal without damaging these organs (in the diseased kidney
is even without effect and, possibly, harmful). In England balsam
resin of copaiba are much employed (1 or 2 grams daily in capsul
Potent diuretics which enjoy a reputation in hepatic cirrhosis are diur
(4 to 6 grams daily), agurin (1 gram three or four times daily), theopl
lin (0.2 to 0.3 gram three or four times daily), theocin, the double
of caffein (from four to six powders daily, each 0.2 gram), and, ab
all, calomel. This last remedy calls for a more minute description.
advise its employment only when the kidneys are normal. Then 0.2 c
gram of calomel are given three times daily for three days, followed
a pause of several days. The oral cavity must be carefully watched. W
marked diarrhea, opium should be given at the same time. I am af
of calomel, which occasionally gives such excellent results, because
stomatitis which, notwithstanding the greatest care, sometimes follows,
debilitate the patient extremely. Its specific action upon the proces
the liver, which is presumed by some authorities, has been nowise pro
Perhaps smaller doses (0.02 to 0.06, five or six times daily), which
specially advised by Saccharjin in hypertrophic hepatic cirrhosis, would
be of value here.

Spa treatment of the affection under discussion at Carlsbad, Ma
bad and Kissingen, etc., is highly recommended, and sometimes als
employment of artificial Carlsbad salt. But a specially beneficial a
upon the liver and the processes going on there these salts cannot
even when given by the mouth (54) in large doses they do not ente
bile. Hence the value of alkalies can only lie in their favorable effect
the gastrointestinal catarrh. Symptoms referable to the gastrointe
tract are often very prominent in these patients, and primarily de
treatment, which, unfortunately, is often without result. Sometimes
symptoms point unmistakably to gastric catarrh (pain in the epigas
anorexia, nausea, vomiting), at other times to involvement of the int
which is usually manifested by uncontrollable diarrhea, more rar
obstinate constipation. Disturbance of the circulation of the blood

portal vessels is certainly the cause of these conditions. In this case the symptoms of gastrointestinal catarrh, which are prominent, call for relief. Here I must refer to the special treatment of these affections, and shall only mention that the spa treatment is very beneficial (besides the springs mentioned above also the waters of Vichy, Neuenahr, Ems, Marienbad, Kissingen, etc., as well as the salt springs of Homburg, Wiesbaden, etc.), especially when constipation and meteorism exist. The stagnant, decomposing masses are discharged, and absorption of the freshly digested foods is promoted, hence the action of the toxic, deleterious products of intestinal bacteria (toxins, ptomains) is limited. In regard to the deficient antitoxic function of the liver, the French (Bouchard, Roger, Dujardin-Beaumetz (55), and others) assign the first place in treatment to "intestinal antisepsis." Charcoal, bismuth salicylate, salol, resorcin, naphthalin, sodium salicylate or sodium benzoate, etc., hydrochloric acid (with subacidity of the stomach), have all been warmly advocated by them, and are frequently employed combined with a diet which will soon be described in detail. So far as we know, antisepsis is carried out in the stomach and the upper portions of the intestine by hydrochloric acid; in the lower portions of the small intestine possibly (?) by the bile acids. It is well known that in advanced hepatic cirrhosis, even when there is little jaundice, only small quantities of bile flow into the intestine, consequently only small amounts of biliary acids find passage there. Connective tissue proliferation in the liver may be enormous, and the greater portion of the hepatic cells may be destroyed, so that microscopically but scant remains of these, which, apparently, are in great part atrophic, are to be found. Deficiency in bile formation and the resulting incomplete antisepsis in the intestine become more pronounced, and the antitoxic property of the liver, on account of the few undamaged hepatic cells which remain, is exerted insufficiently. In the course of hepatic cirrhosis we by no means rarely meet with conditions which point to autointoxication of the organism with these intestinal toxins. These conditions have already been explicitly described (56).

In Germany, quite correctly, the theory of "intestinal antisepsis" has met with little acceptance. The extremely artificial superstructure erected in France lacks a sufficiently firm foundation, the entrance is not free from danger, and it may very readily and suddenly collapse. Preliminary studies upon which to found this treatment must be characterized as entirely inadequate. I am convinced that, in the disease in question, as little headway has been made with intestinal antisepsis as, for example, in enteric fever, which in France is treated according to similar principles. The administration of small doses of antiseptics is useless, large doses are decidedly dangerous. A combination of various antiseptic remedies (of each almost its toxic dose) has given no better results. We will do well to continue to be skeptical in regard to this therapy, and to rely upon purgatives such as aloes, senna, calomel, rhubarb (Heim's pills have been most tensively used in the treatment of hepatic cirrhosis), and the laxative b waters. It is simply impossible to disinfect for years the intestine human being, all the more so since we do not know which bacteria sh be destroyed, and which toxins should be removed. Continued resear here necessary, and then perhaps, but only perhaps, treatment of this may be successful.

A chronic disease, the course of which, like the one in question, prolonged, naturally necessitates care in the diet, and here the pati food must be simple and non-irritating with a sufficient amount of calo It should be composed of milk, carbohydrates in the form of wheat b crackers, flour, zwieback, mashed potatoes, cocoa, and rice, with the tion of butter and meat. Only the more easily digested and nutri vegetables, such as green peas, turnips, asparagus, cauliflower, and spi are to be allowed, while, on the other hand, those which cause distenti such as all kinds of cabbage—are to be avoided.

As an ideal diet in hepatic cirrhosis the French have praised and tically employed one strictly of vegetables. The guiding thought is t that in its employment the formation of toxins (from meat) is suppre and intestinal antisepsis is facilitated. In carrying out such dietetic r the utmost care is necessary. Few patients with hepatic cirrhosis adhere to such a diet on account of their especial tendency to gastric intestinal ailments. For individual points in the diet, I must refe authoritative text-books (57).

Surgical Measures.—To facilitate the establishment of the above tioned collateral circulation between the portal vein and the veins o body, that is, to make it possible, and thus relieve the dangerous c quences of congestion in the portal system, efforts have been made, fo ing Talma's (57 a) advice, to fasten the omentum, and also the s to the abdominal wall. In this manner adhesions containing new v are formed, and may bring about the desired condition. This operat not always successful; disappointment and even serious results (gan of the omentum, infections) have followed; but, on the other hand, times results which are favorable. In some cases, after the operatio ascites was checked, and the patients left the hospital in the best con and able to carry on their occupations (Talma, Drummond and M Neumann (57 b)). Such favorable results as these from Talma's tion justify us, in suitable cases, in resorting to it. With our inc experience regarding the operation itself, and in the choice of s cases, we can hardly doubt that in the future these results will b more gratifying. Experimental investigations (Tillmann (57 c)) c the practicability and the benefits of Talma's operation; compreh reviews by Friedmann (57 d) and Rohmer (57 e) give critical rep numerous cases operated upon, and a recent publication by Le may be referred to (*Deutsche med. Wochenschr.*, 1903, Nr. 48, p. 843) in which suggestions have been given regarding the choice of cases to be operated upon.

C. HYPERTROPHIC HEPATIC CIRRHOSIS

PATHOLOGICAL ANATOMY

In important points this affection differs pathologico-anatomically as well as clinically from atrophic hepatic cirrhosis, and therefore, as is now generally admitted, is entitled to a special position.

Up to the time of death the liver usually remains greatly enlarged. The surface is tumid, it is of firm, elastic consistence, its color strongly icteroid, yellow to green. Upon section a decided increase of connective tissue is seen; this is not only found in the interlobular spaces, but also in the hepatic lobules themselves. In spite of the enormous increase of the connective tissue, the mass of the hepatic parenchyma is unaltered. The connective tissue does not contract, the liver cells are not destroyed; on the contrary, according to some authors (Ackermann (58), Aufrecht) they are enlarged, hypertrophic, and mostly polynuclear. That the connective tissue proliferation in hypertrophic hepatic cirrhosis takes place especially around the biliary passages and, according to the view of the French, chiefly Charcot and his pupils, originates from these and not from the blood-vessels, has by no means been proven, and has been denied by me for many years (*Verhandlungen des Congresses für innere Medicin*, 1892).

Since the finer biliary passages and the blood-vessels coalesce in the same interstitia, it is hardly possible from the results of microscopic investigation to maintain with any degree of positiveness that the one system, and not the other, is implicated in the connective tissue increase. This is not contradicted by the fact that, in many areas, connective tissue proliferation is greatly developed, particularly around the biliary passages, that their walls are thickened while the lumen remains open. The biliary channels, on account of their structure, form a prominent part of the connective tissue masses. The incorrect view of the French is, as was previously remarked, based upon the fact that they do not distinguish "biliary cirrhosis," which we shall specially describe later, from hypertrophic cirrhosis, and this has caused much confusion. So-called, newly formed biliary passages are more numerous than in the atrophic form. It must be expressly stated that the differences between these two forms are only marked when we compare well-developed classical cases, that these, however, are by no means always typical, and that these forms merge into one another. Thus, in hypertrophic hepatic cirrhosis there is sometimes contraction, atrophy of the cells, etc. The French have easily surmounted these difficulties by creating their "forme mixte." But this, at the s time, indicates that special differences between atrophic and hypertro hepatic cirrhosis do not exist. The pathologic anatomy of the dis which apparently occurs in France much more frequently than in Germ has been particularly studied by the French—I mention only such na as Charcot (59), Ollivier (60), Hayem (61), Gombault (62), Hanot (Schachmann (64) and, among German authors, besides Ackermann (*l.* Rosenstein (65) and Stadelmann (66). In France this form of hep cirrhosis is named after the most eminent authority in the realm of disease, Hanot's disease. After all is said, we must frankly admit the true cause of differences, even of a pathologico-anatomical nature, tween atrophic and hypertrophic hepatic cirrhosis is unknown. It is wo of mention that, occasionally, even well-developed cases of hypertro hepatic cirrhosis cannot be recognized macroscopically, but are only termined by microscopic examination. This is due to the peculiar, but only slightly prominent, structure of the richly developed conne tissue, and the absence of contraction.

SYMPTOMATOLOGY

The onset of the disease, like atrophic hepatic cirrhosis, is accompa by indefinite symptoms on the part of the gastrointestinal tract. patient complains of anorexia, vomiting, pressure in the epigastrium, stipation, and diarrhea. A very intense jaundice soon appears, so this form of the affection has been designated in France as "cirrhose hy trophique avec ictère." The liver is greatly enlarged, is coarse and h with a rounded margin, and remains enlarged to the end. The jaun may disappear in the early stages of the disease but may several ti recur in paroxysms until it finally becomes permanent. Besides enla ment of the liver, we usually find a very marked enlargement of the spl Ascites is almost invariably absent. There is a marked tendency to he rhage. In spite of the jaundice, the feces usually show bile. Death o from exhaustion after a prolonged duration of the disease, which ru longer course than atrophic hepatic cirrhosis (four, five, ten to t years), and is frequently preceded by the symptoms of autointoxic and by cerebral phenomena. Irregular rises in temperature are not The cause of the enlargement of the spleen is doubtful; it cann congestion, as in atrophic hepatic cirrhosis, for this is entirely absent the clinical picture. Nothing remains but the assumption of a p in the spleen resembling that taking place in the liver. Rosenstei mentioned a decrease of the red blood-corpuscles.

Disease of the kidney is rarer than in atrophic cirrhosis.

As *complications* there are to be mentioned general and local tonitis (perihepatitis, perisplenitis), as well as myocarditis.

ETIOLOGY

Hypertrophic hepatic cirrhosis, similar to the atrophic form, occurs chiefly in men, less frequently in women. It is quite a rare disease, and is apparently restricted to certain localities. Thus, in France and Holland (Rosenstein) it is comparatively frequent, while in Germany, at least in its typical forms, it is very rare. Most authors attribute the affection to alcohol, others contradict this (Rosenstein); it is certainly very remarkable that, notwithstanding the plentiful ingestion of alcohol in Germany, the affection is there so rare, while, in other regions, in spite of the same conditions, the disease is often noted. This forces us to the conclusion that alcohol cannot be the only cause, but that an unknown factor is added. It has also been assumed that it is due to a parasitic disease of the biliary channels (bacteria, protozoa). Positive proof of this is lacking.

DIAGNOSIS AND PROGNOSIS

In the stage of onset the affection is just as difficult to recognize as atrophic hepatic cirrhosis. The gastrointestinal symptoms are not characteristic, the jaundice may be purely catarrhal, and is the more likely to be considered such because this symptom, as well as the enlargement of the liver, often decreases. Only with the permanent enlargement of the liver, the decided splenic tumor, the persistent jaundice, does the diagnosis, until then tentative, become positive.

Prognosis.—The prognosis is unfavorable. Recovery from the well-developed disease is unknown; that the malady may be arrested, or even cured, in the stage of onset by suitable treatment is possible, but not proven, since, in this stage, the diagnosis is still uncertain. It is at least a source of comfort to know that the course of the affection may be very prolonged without decidedly interfering with the activity of the patient.

TREATMENT

At the onset of the malady the treatment usually employed in gastro-intestinal affections is to be instituted; the jaundice present, which is generally regarded as catarrhal jaundice, should receive appropriate—also dietetic—treatment. The effects of the abuse of alcohol must be combated, and its future avoidance always be insisted upon by the physician.

In the case of hypertrophic hepatic cirrhosis the employment of calomel in fractional doses, as advised by Saccharjin (0.02–0.05, four or five times daily), is particularly valuable. According to this author, the drug has a specific action and brings about a cure. But that a specific action upon the process in the liver is not produced by the use of calomel, and that the expected result frequently does not follow, must be especially emphasized. Nevertheless, with all necessary precautions (thorough care of the mouth, calomel for three or four days, then a pause of three or four days, then another calomel period, etc., the treatment to be kept up for about four weeks), the calomel treatment is advisable. Potassium iodid should also be tried in hypertrophic hepatic cirrhosis, although I have seen little success from its employment, nor have I found such reported in literature. Ebstein (66) maintains that he has had good results with potassium iodid.

The disturbances of metabolism in the hypertrophic form do not differ from those in the atrophic. Absorption and the assimilation of food show no deviation except that, perhaps, on account of the deficiency of bile in the feces and the jaundice present, the absorption and assimilation of fats in the intestine are disturbed. The foundation for nutrition is milk, to which carbohydrates and proteids should be added as required. Details may be found in text-books on dietetics (52).

D. BILIARY HEPATIC CIRRHOSIS

In France the disease under discussion is usually grouped with hypertrophic hepatic cirrhosis, as has already been mentioned, and this has caused great confusion. We consider it advisable sharply to differentiate the two conditions, and in Germany, so far as I can see, we designate by this term the cirrhosis which arises in connection with mechanical closure of the bile channels, especially the common bile duct, either by stone, by a tumor, by adhesions, by cicatrices, etc.

ETIOLOGY AND PATHOLOGY

After prolonged biliary stasis (Janowski) necrotic foci due to destroyed hepatic cells are found in the liver. These are absorbed, small cell infiltration appears about the surrounding tissue, and, later, connective tissue proliferation develops. The bile channels are also thickened by infiltration and connective tissue formation. New bile channels are formed in the same manner as in ordinary hepatic cirrhosis. The connective tissue increase, as well as the atrophy of the parenchymatous cells, may finally attain the extreme proportions seen in atrophic hepatic cirrhosis. Consequently, as in the other affection, the picture of portal vein stasis appears, and in this stage biliary cirrhosis can no longer be differentiated from cirrhosis due to other causes (Litten (67), Mangelsdorf (68)). We then find fibrinous connective tissue bands which constrict and permeate the acini, connective tissue proliferation around the portal vein branches which become thickened and impermeable, and newly formed biliary channels, etc.

Experimental investigation, in the main, agrees with observations in man, but the consequences of experimental biliary stasis are much less prominent in the liver. Infection with its consequences (purulent inflammation, abscess) may complicate the situation in the animal experiment, and it is not always absent in man.

SYMPTOMS

The affection runs its course under the picture of severe jaundice, of cholemia, that is, of autointoxication, and usually with such rapidity that the severe hepatic changes above described do not have time to develop completely. The disease rarely lasts longer than two years, and, by its more rapid course, as well as by the absence of decided enlargement of the spleen, biliary hepatic cirrhosis differs from hypertrophic hepatic cirrhosis to which it otherwise presents great similarity, especially if the liver is greatly enlarged, which is often the case. In the differential diagnosis the observation of the feces is important. In hypertrophic hepatic cirrhosis they are more or less bile stained; but in biliary cirrhosis they are acholic, and in the latter affection the icterus is usually more intense. Ascites, as well as the other signs of portal vein stasis (also enlargement of the spleen), may develop in the terminal period (Janowski (69)).

TREATMENT

The treatment depends, in the main, upon the etiology. As the affection always depends upon mechanical causes which cannot be removed by internal treatment, in biliary cirrhosis we are therefore compelled to resort to surgical measures. Stones must be removed by operation, and, in the case of cicatricial adhesions and tumors which cannot be removed, cholecystoenterostomy is to be performed, that is, the gall-bladder is to be united with the intestine. This operation seems to be too rarely performed, and we cannot sufficiently emphasize that it is calculated to save the life of many a patient, or, at least, decidedly to prolong it, and gives us fresh hope.

The greatest attention must be paid to the nutrition, and, in the main, the same laws are operative as in hypertrophic hepatic cirrhosis. Carbohydrates and proteids are preferable, while the fats should be as far as possible excluded, since, in acholia of the intestine, fats are insufficiently absorbed; on the other hand, milk should be employed to the fullest extent.

E. SYPHILIS OF THE LIVER

That syphilis of the liver produces interstitial hepatitis may be regarded as certain; nevertheless, one and the same poison, as must be admitted, may produce such different forms of disease of the liver that we cannot regard it as strange when we see the same conditions as in the case of alcohol.

The connection between syphilis and hepatic cirrhosis is not always easy to recognize, and often can only be determined late, provided the history of our patients does not furnish the clue; for, clinically, the stage of onset of syphilitic hepatic cirrhosis very rarely differs from that of the alcoholic form. In syphilitic hepatic cirrhosis the development nective tissue rich in cells takes place, and this has a tendency to r cicatricial contraction, so that the tissue later becomes tough and fi and the cellular elements disappear. The parenchyma of the liver is destroyed, and fatty degeneration and atrophy occur in the hepatic c It is quite necessary, too, to differentiate congenital syphilitic hepa cirrhosis from acquired hepatic syphilis.

The first is found in children with congenital syphilis, and here t above mentioned characteristics of *diffuse* hepatic cirrhosis are most e dent. The liver is distinctly enlarged, tough, brownish, with many sig of new and old connective tissue in the interlobular structure, and t may also permeate the lobules. The blood-vessels are greatly thicken their walls show cellular infiltration. But, besides the diffuse connect tissue increase which presents the greatest similarity to that of ordin hepatic cirrhosis, there are massive connective tissue strands in individ areas and foci with an accumulation of small cells.

In such children the liver is usually hypertrophic, probably beca these children early succumb to the underlying affection (syphilis and consequences), that is, before the atrophy which is observed in some c that live longer has time to develop. The liver is sometimes smo sometimes flabby and notched, sometimes also coarse and granular. sides the diffuse disease of the organ, we find—however, more rarely formation of gummata in the form of small nodules distributed over entire surface. The process in the liver strongly resembles syphilitic ease of other organs. The disease without doubt begins during the f period; the fetus is often decomposed, or the infants die a few w or months after birth. Syphilis of the liver is the most frequent pl of hereditary syphilis.

Hepatic syphilis of adults is a late phenomenon of acquired syph appearing in the tertiary stage, and may be divided into two differ forms, which, however, may also be combined.

1. The diffuse form corresponds to syphilitic cirrhosis of children to alcoholic cirrhosis, and neither pathologico-anatomically nor clinic can it be positively distinguished from the latter. Syphilis and alco are usually combined as deleterious factors in one and the same patient

2. Syphiloma of the liver, that is, gummatous hepatitis. Gummat nodules are characteristic of this form; they vary in size from that o nut to that of an apple, are mostly found upon the concave surface of liver near the suspensory ligament, but may also be noted at the po hepatis in Glisson's capsule. These nodules show a tendency to caseat to decay, and, in connection with this, to contraction. Then deep furr develop and cause constriction of the liver, hence the organ may be s rated into individual lobes ("lobulated liver"); these constrictions sist of hard, tough connective tissue, perhaps also of remains of a case

gummatous tissue, and sometimes they contain unchanged gummatous nodules. Upon the smaller and larger blood-vessels (hepatic arteries, portal vein) connective tissue proliferation is frequently found in the form of syphilitic endarteritis. The serosa of the liver is usually thickened in consequence of inflammatory processes which may also cause extensive adhesions to the neighboring organs.

Finally, in hepatic syphilis of adults, as in that of children, the syphilomata may be of such a miliary form that a certain resemblance to tubercle is noted. In adults gumma formation, and in children the diffuse development of connective tissue, is typical of syphilis of the liver. But in adults other symptoms of tertiary syphilis are usually present (testicles, meninges, bones, mucous membranes, etc.).

SYMPTOMS AND CLINICAL COURSE

Syphilitic hepatic cirrhosis, at least so far as the gummata are concerned, usually runs its course without symptoms, and is frequently discovered only at autopsy. In inflammation of the serosa, if adhesions form, pain in the hepatic region is not a rare complaint. Frequently, however, the cicatricial retractions, the lobulation of the liver, as well as the large gummata, may be recognized at the examination, but the latter by their great nodular prominences often lead to errors in diagnosis by being mistaken for malignant tumors.

If the gummata and the cicatrization are situated at the porta hepatis, and form a constriction in this area, the well known symptoms of stasis, even icterus gravis, may arise in consequence of closure of the common bile duct.

[Gummatous hepatitis frequently produces an irregular type of fever. This is sometimes manifested by chills, fever and sweats occurring at irregular intervals and at other times by a moderate fever of intermittent or remittent type without chill. Frequently the liver is palpable, its consistency increased, and it is tender to the palpating hand during the febrile attacks. Rest without medication results in relief in some instances. The recognition of the cause with appropriate treatment with mercury and iodids is followed by entire relief.—Ed.]

As the liver is otherwise well able to perform its functions, and diffuse processes are rare in the adult, as has been mentioned, the constitutional condition is in most cases not affected, nor is life endangered, by the disease. Only when diffuse syphilitic hepatitis develops is the entire clinical picture of atrophic hepatic cirrhosis presented.

Amelioration and arrest of the affection, particularly under proper treatment, is very frequent.

Amyloid degeneration of the liver, spleen, and kidneys may follow, as n any cachexia; in severe syphilis this is not unusual, and it may then oth complicate and disguise the clinical picture.

In children, usually in the new-born, the inverse is the case. The children are atrophic, cachectic, and do not thrive; ascites, enlargement and all of the other symptoms of diffuse hepatitis develop rapidly, and the children, as a rule, soon succumb to the disease. In milder cases, however, the affection may last for years. Amyloid degeneration of the liver and of other organs is then a frequent complication. The children, even the mild cases, rarely reach puberty, they have no vigor, they develop slowly, and remain weak and thin—just as is commonly observed in syphilis hereditaria tarda.

DIAGNOSIS

The diagnosis of syphilitic disease of the liver in young children is usually easy. The history of the parents, the presence of other symptoms of hereditary syphilis, and the examination of the children, soon make the diagnosis positive. The distended abdomen, the enlarged, hard liver which is readily palpable, the large spleen, the symptoms of congestion, ascites, etc., and the absence of other etiology pointing to hepatic cirrhosis, reveal the true nature of the malady even when the parents falsify their history during the examination, and conceal their own syphilis.

In adults, confusion with alcoholic cirrhosis on the one hand, and malignant tumors of the liver on the other hand, is easily possible. Even without a history, other signs of tertiary syphilis, such as cutaneous ulceration, enlargement of the bones, and ulceration in the throat, point with certainty to the correct conclusion. A complicating amyloid degeneration should be remembered, but should not lead us astray. If there is no other reason for its existence, in doubtful cases it favors syphilis. In a differentio-diagnostic respect, the slight cachexia may be considered as against carcinoma.

As contra-indications of alcoholic cirrhosis there are varying symptoms —longer-continued, spontaneous improvement, the enormous spleen, the size of the nodules, the marked globulation of the liver, and the possible absence of alcoholism, etc.

Confusion with simple corset lobe liver is easily possible. But this almost always occurs in women, and the furrow from lacing is a single one, although it may become very deep and almost divide the liver into two parts, while in syphilitic hepatitis there are several cicatricial bands, therefore lobulation occurs in several areas of the organ.

TREATMENT

With early and proper specific treatment, the disease may be arrested, the process retarded, and even a relative cure take place. The etiology and an early diagnosis are, therefore, of vital importance for the patient. Cicatricial constriction can never be removed, and, according to our present experience, it may be regarded as more than doubtful that a well-developed diffuse process can be cured or even arrested.

Nevertheless, when syphilis has been determined and disease of the liver has appeared, a careful antisyphilitic treatment with potassium iodid and mercury is not only invariably advisable but directly indicated, and this treatment should be instituted even when the diagnosis of syphilitic hepatic disease is doubtful. Frequently, as is not unusual in syphilis, the result of treatment confirms the diagnosis. Cautious treatment with potassium iodid and mercury is not injurious if the patient's kidneys are normal and he is continuously under professional observation. The physician should, therefore, first try potassium iodid or sodium iodid in increasing doses, so that after a time several grams of the iodin salt will be given daily, and in combination with this, as soon as we see that the iodin has had its effect and the condition of the patient will permit it, treatment with mercury should be begun (preferably a thorough inunction cure). Decided pain in the hepatic region is the consequence of perihepatitis, and may justify such procedures.

The other symptoms of hepatic disease are to be treated in accordance with the method established for the treatment of atrophic and hypertrophic hepatic cirrhosis.

LITERATURE

(1) *Ackermann, Virchow's Archiv,* LXXX.
(2) *Hartung,* "Ueber das histologische Verhalten der Leberzellen bei der Alkoholcirrhose." *Dissertation,* Halle, 1889.
(3) *L. Brieger, Virchow's Archiv,* LXXV.
(4) *Virchow's Archiv,* LXII.
(5) *Arch. gén. de Méd.,* 1885.
(6) *Deutsches Arch. f. klin. Med.,* 1895, LV.
(7) *Zeitschr. f. klin. Med.,* 1896, XXIX.
(8) *Deutsches Arch. f. klin. Med.,* XXXIV.
(9) *Thèse de Paris,* 1888.
(10) *Arch. gén. de Méd.,* 1889, p. 513.
(11) *Archives de Physiologie,* 1878; "Maladies des pays chauds," Paris, 1889.
(12) Quoted from Quincke, "Die Krankheiten der Leber," p. 420.
(13) *Revue de Méd.,* 1882.
(14) *Festschrift für Albrecht v. Haller,* 1877.
(15) *Arch. de physiol. norm. et path.,* 1886.
(16) *Archivio medico italiano,* 1882.
(17) *Arch. gén. de Méd.,* 1882.
(18) *Berliner klin. Wochenschr.,* 1890; *Verhandl. d. Congr. f. innere Med.,* 1892.
(19) Quoted from Ewald in *Eulenburg's Realencyclopädie,* XI.
(20) *Deutsches Arch. f. klin. Med.,* XXXI.
(20a) *Berliner klin. Wochenschr.,* 1890.
(21) *Arch. gén. de Méd.,* 1888.
(22) *Fortschr. d. Med.,* 1884.

(23) *Vierteljahrschr. f. gerichtl. Med.*, LXXXVIII.
(24) *Ziegler* und *Nauwerk, Beiträge*, etc., IX, Heft 2.
(25) *Ziegler* und *Nauwerk, Beiträge*, etc., VIII.
(26) *Arch. de Pharmacodynamie*, 1895, II.
(27) *Thèse de Paris*, 1894, quoted from Quincke.
(28) *Virchow's Archiv*, CX.
(29) *Ziegler's Beiträge*, II.
(30) *Deutsches Arch. f. klin. Med.*, XXIII und LVIII.
(31) *Thèse, Paris*, 1879.
(32) *Deutsches Arch. f. klin. Med.*, 1882, XXX.
(33) *Journ. de Méd.*, Bruxelles, 1877.
(34) *Arch. des sciences physiques et naturelles*, Genève, 1877.
(35) *Philadelphia Med. Times*, 1877.
(36) "Leçons sur les autointoxications," 1887.
(37) "Action du foie sur les poisons." *Thèse de Paris*, 1887.
(38) *Arch. f. experim. Path. u. Pharm.*, XII.
(39) *Deutsches Arch. f. klin. Med.*, 1883, XXXIII.
(40) *Deutsches Arch. f. klin. Med.*, 1889, XLV.
(41) *Zeitschr. f. physiol. Chemie*, XVII.
(42) "Lehrbuch der Pathologie des Stoffwechsels," 1893.
(43) *Arch. f. experim. Path. u. Pharm.*, XXXIII.
(44) *Arch. f. Physiol.*, 1891, II.
(45) *Verhandl. d. XI. Congr. f. innere Med.*, 1892.
(46) *Morgagni*, 1897.
(47) *Arch. f. experim. Path. u. Pharm.*, 1892, XXXII.
(48) *Zeitschr. f. klin. Med.*, XLVII.
(49) *Deutsches Arch. f. klin. Med.*, LXXIV.
(50) *Deutsche med. Wochenschr.*, 1901.
(51) *Semaine méd.*, 1901; *Revue de méd.*, XXI.
(52) *Berliner klin. Wochenschr.*, 1901, Nr. 18.
(53) *Münchener med. Wochenschr.*, 1903, Nr. 8.
(54) "Untersuchungen von Dr. Glass aus meinem Laboratorium," *Arch. experim. Path. u. Pharm.*, XLIII.
(55) "Traitement des maladies du foie," Paris, 1893.
(56) Compare also *Stadelmann*, "Ueber chronische Leberentzündung." V *handl. d. XI. Congr. f. innere Med.*, Leipzig, 1892, and *Stadelma* "Der Icterus und seine verschiedenen Formen," Stuttgart, 1891.
(57) *Stadelmann*, "Die Ernährungstherapie bei Leberkrankheiten" in v. Leyde "Handbuch der Ernährungstherapie," II.
(57a) *Berliner klin. Wochenschr.*, 1898, Nr. 38, u. 1900, Nr. 31.
(57b) *Deutsche med. Wochenschr.*, 1899, Nr. 26.
(57c) *Deutsche med. Wochenschr.*, 1899, Nr. 18.
(57d) *Centralbl. f. d. Grenzgebiete d. Med. u. Chir.*, 1900.
(57e) *Deutsche Aerzte-Zeitung*, 1901, Heft 14–15.
(58) *Virchow's Archiv*, LXXX.
(59) "Maladies du foie," 1877.
(60) *L'Union méd.*, 1871.
(61) *Arch. de physiol.*, 1874.

(62) *Arch. de physiol.*, 1876.
(63) *Thèse de Paris*, 1876; *Arch. gén. de méd.*, 1877–79.
(64) *Arch. de physiol.*, 1887.
(65) *Rosenstein, Stadelmann, Verhandl. d. XI. Congr. f. innere Med.*, 1892.
(66) *Verhandl. d. XI. Congr. f. innere Med.*, 1892, p. 128.
(67) *Charité-Annalen*, 1880.
(68) *Deutsches Arch. f. klin. Med.*, 1882, XXXI.
(69) *Ziegler's* "Beiträge zur pathologischen Anatomie," 1892.

NEOPLASMS OF THE LIVER AND BILIARY PASSAGES

By FR. KRAUS, Graz

The development of the surgery of the liver and gall-bladder in th last few decades, gradual at first but lately proceeding with more rapi strides, has steadily intensified the practical interest of the profession i neoplasms of the liver. There are surgeons who dream of the complet eradication and actual cure of these diseases, even of malignant growth by the aid of a diagnostic exploratory incision. Even more may, perhap be expected on account of the positive etiologic relation of carefully chos cases of carcinoma of the biliary passages (perhaps also of the liver) cholelithiasis if the operative treatment of gall-stone disease is resorted early enough. If, however, the knife can actually aid us, and to avert t dangers in general of cholelithiasis, of which lithogenous cancer is or *one,* a very limited but, as I believe, sufficient experience has taught that in permanent jaundice which is the result of an already present c cinoma of the common bile duct, operative interference is of comparativ little use, and even exploratory laparotomy, in individuals whose comm bile duct is occluded by a malignant tumor, is very badly borne. T danger involved, therefore, in the operation for gall-stones is one of practical reasons why so much depends upon a knowledge of diagno that is, an exact recognition of the factors which bring about chr obstruction of the common bile duct by concrements and by neoplasm the biliary passages themselves, of the duodenum, or of the head of pancreas, etc., enables us to differentiate them.

The very complex clinical picture occasionally makes the decisive di entiation of malignant neoplasms of the liver itself from those of gall-bladder and of the biliary passages very difficult, as primary ca of the gall-bladder proliferating into the liver there reaches its gre development, and as all carcinomata which occlude the duodenal en the common bile duct, no matter what their origin, give rise to metas in the liver, etc. The simultaneous description of these various neopl therefore, appears to me to clear the situation but little, and after consideration of the varying practical indications, I believe a separat scription of this subject to be necessary. On account of the prev opinions concerning carcinoma of the liver as a primary or seco affection, its interesting histogenetic development, its manifold varieties, and probably also because of considerations to which the system of tumors of the liver gives rise, purely clinical interest in the latter is far less; hence the following order of arrangement will be different.

As indicated, we shall discuss manifold clinical pictures, but the combination of the following guiding symptoms will always be observed: General digestive disturbances, and, in connection with these, rapid and unarrested progressive cachexia, the appearance or the absence of a tumor, metastases in the liver itself or elsewhere, cirrhosis of the liver (secondary), compression of the biliary passages, particularly that form accompanied by permanent jaundice, portal vein stasis.

1. NEOPLASMS OF THE GALL-BLADDER

As a type of tumor of the gall-bladder *carcinoma* may be considered, for the other forms of tumor mentioned in literature are either so rare or of such subordinate importance as to be of only slight interest to the physician. *Sarcoma* has the same significance as carcinoma.

Among the granulation tumors of the gall-bladder, *tubercle* must be mentioned (Lanceraux, Heddaeus, Mermann); of benign tumors there are to be considered *true cystic tumors* (Wiedemann), *mucus polypi, submucous fibromata* (Albers), *papillomata* (Riedel, Kümmel), *lipomatous* structures (subperitoneal upon the gall-bladder, Cornil-Ranvier), *aneurysm* of the cystic artery opening into the gall-bladder (Chiari), *echinococcus*; finally, of other malignant growths we must enumerate papillary *myxomata* (Schüppel), *sarcomata* (Czerny, Riedel, Destrée, etc.).

Cancer of the gall-bladder is a pathologic condition which has only recently been accurately studied. Early descriptions were given by Stoll, Hall, Baillie, Heyfelder and others (1777–1839). The first positive cases were probably described by Durand-Fardel (1837–1840). Villard (1871), in his "Étude sur le cancer primitif des voies biliaires," collected 24 cases of primary cancer of the biliary ducts, of which 21 were carcinomatous degeneration of the gall-bladder. In a subsequent series of reports, the most important are those of J. Krauss and H. Zenker, the latter author having most thoroughly and convincingly described as the most important factor in the etiology of cancer of the gall-bladder the presence of gall-stones. Noteworthy anatomical indications, in addition to those already mentioned, are contained in a publication by Ohloff. The most comprehensive statistics have been compiled by Courvoisier and Musser. Among recent comprehensive publications from a surgical standpoint, that of A. Heddaeus must be mentioned. At the present time, the pathologic anatomy and the symptomatology of cancer of the gall-bladder are well understood, and only a few questions touching the border-line between internal medicine and surgery call for further explanation.

Cancer of the gall-bladder is only very rarely secondary, that is, proliferating into the gall-bladder from neighboring organs (the pancreas, the duodenum, the stomach, etc.). The earlier view (Baillie), according to which the gall-bladder was said to be most rarely attacked by cancer except when the disease had previously developed in the liver, has bee entirely abandoned. There are numerous cases on record in which th gall-bladder was the only organ which showed malignant involvement, th liver being entirely free. And this is favored by the fact that canc of the liver which occurs simultaneously has, as a rule, proliferated l contiguity, or is to be looked upon as metastasis of the gall-bladder. I old complicated cases with disease extending to all the organs in the regi of the liver, at the present time the gall-bladder is always assumed to l the primary seat if it show but a moderately severe implication. In su cases we occasionally find in the liver a tumor mass with indefinite, limit borders, embedded in cicatricial adhesions, in the center of which up incision a cavity is disclosed with irregular borders and a pappy or pur lent mass containing concrements. The carcinoma proliferating from tl gall-bladder to the adjacent liver naturally gives rise to a tumor whic at first sight, may resemble a primary massive cancer of the liver. On in one case, Tyson's, was the carcinoma of the gall-bladder of pure metastatic origin, and this growth was found at the autopsy in a ca in which three operations for cancer of the lip had been performed.

As a rule, cancer of the gall-bladder arises primarily either from t fundus or the neck, rarely from the middle portion, or the wall adjace to the liver. It occurs either as scirrhus or adenocarcinoma. The s cancerous alveoli readily break down and form a malignant ulcer. Collo cancer (Villard, Aezel, Biach, Roberts, Janeway, Brodowski) and villo cancer (Heschl, Klob, Moxon, Voss, Fifield) occur. Conspicuous, y particularly mentioned only by Mulat, is the pavement cell epithelium wi the well known onion-like microscopic configuration. Either the canc surrounds the gall-bladder, and only implicates a small portion of the wal or the entire wall is uniformly infiltrated. Often there is but a sm tumor, which nevertheless has become the focus for a large, seconda cancer of the liver, or has produced extensive metastases. The enti malignant gall-bladder has contracted upon one or a few gall-stones. I other cases the gall-bladder appears as a nodular tumor of the size of fist. Occasionally at its fundus it forms a diverticulum which is separat from the rest of the organ by a thickened ring and contains gall-stone this ring is usually the seat of the cancerous development. As is obvio from the foregoing, the carcinoma usually proliferates to the surroundi areas. Widely extended dendritic infiltration of Glisson's capsule is r rare. In particular, the adjacent liver, the parietal peritoneum, the om tum and the intestine (above all, the colon) are primarily implicated pericystitic adhesions, and the secondary growth penetrates the connec ue membranes. Extending by way of the hepato-duodenal ligaments cancer reaches the portal lymph nodes, which become markedly en- d, and by pressure upon the portal vein produce ascites. Thrombosis e trunk or of the larger branches of the portal vein is not rare. By uity through the cystic duct the neoplasm attacks the common gall- nd the hepatic duct, and by stenosis causes obstruction to the outflow e, dilatation of the gall-bladder, and stasis jaundice. The hepatic nd common gall-duct may also be implicated externally by the car- a. In some cases a further distribution into the small intra-hepatic ucts has been observed, so that upon transverse section they appear cerous rings. This gives rise to the formation of new bile passages trophy in the trabeculæ of the cells of the liver acini, causing the ion of bile in the liver and the formation of small cysts which are d with the epithelium of the biliary passages. In the further course affection, inflammatory products collect. The malignant mass breaks upon the inner surface, the tissue threads reach the bile, which also poses, and is changed into a dirty, grayish-brown fluid admixed with and pus. The process of destruction usually extends even further, adjacent tumor masses between the stomach, the large intestine and er, and thus an ichorous cavity develops which is filled with a dirty, mass, permeated with cancerous nodules. Continued stasis of bile onsequent infection produce a distributed cholangitis. Not rarely s form which perforate into the liver, into the gastrointestinal canal, ito the abdominal wall (navel).

though cancer of the gall-bladder by continuous proliferation is prone ack the adjacent liver and the biliary passages, as well as quite only the porta hepatis, it may also give rise to *metastases,* as I must eiterate in opposition to the opinion which has been generally main- since the time of Courvoisier. The metastases are not only found liver, but, considering the brief duration of the disease, often in and quite distant organs (in the peritoneum, the omentum, the re- of the umbilicus and the urinary bladder, the recto-vesical fold, the , the spleen, the intestine, the lung, the pleura, the ovaries, the portal, eritoneal, mesenteric, and omental lymph-glands). I know of a case ich an incorrect diagnosis was made because the first and chief ob- symptom consisted of tumors which were found in the pelvis.

ETIOLOGY AND PATHOGENESIS

e *etiology* and *pathogenesis* of carcinoma of the gall-bladder there- ecessitate the most minute investigation on the part of the practi- because of the difficulties which up to the present time have suc- ly resisted a surgical treatment of the affection, and which depend, small degree, upon the fact that the chief cause of cancer of the gall-bladder is not sufficiently considered, and operation is generally deferred until too late. Almost all investigators have been struck by the extraordinary frequency, more than mere coincidence, with which stones (or ulcers and cicatrices due to stone) as well as cancer are found in the gall-bladder. Naturally the question arises, What causal relation exists between the presence of gall-stones and the formation of cancer? While some authors were formerly inclined to regard the development of cancer as primary, for 30 years past there has scarcely been a pathologist who doubted that cancer is due to the irritation of stones that have previously formed (Klob, Frerichs, Klebs, Willigk, Schüppel, Krauss, Zenker, Courvoisier, Naunyn, and others). This connection is, in the first place, borne out by statistics. Courvoisier found gall-stones simultaneously with cancer of the gall-bladder in about 90 per cent. of all cases, Siegert in almost all, and Löbker in all cases. According to Schröder, 14 per cent. of all patients with gall-stones are attacked with carcinoma of the gall-bladder according to Naunyn among 150 such cases, 14 developed cancer of th biliary passages. Of 172 patients with gall-stones operated upon by Löbker 17 suffered from carcinoma of the gall-bladder, and Riedel assumes tha about 10 per cent. of all patients suffering from cholelithiasis subsequentl acquire carcinoma. The presence of stones prior to the development o cancer can be proven clinically as well as pathologico-anatomically. It ha long been known that carcinomata are prone to form in cicatrices, partic larly in the stomach, as has recently been clearly proven by G. Hause and H. Zenker conceived the idea that the development of cancer of t gall-bladder might be similarly connected with preceding ulcerative pro esses which might be attributed to pressure necrosis due to stones. Zenk demonstrated clearly that the successive cicatrices cause an atypical gland lar proliferation which predisposes to carcinoma. The increased grow of the remaining glandular portion, the shooting forth of sprouts a their extension in various directions, the more marked lobular formati are primarily but the expression of a regenerative endeavor of the tissu the character of the excessive and atypical growths gains prominence by chronic irritation of the stones. Hauser also considers the absence physiologic resistance, and Ribbert the constriction of portions of the gla by connective tissue, as important. Ohloff's conception is somewhat ferent; he believes the metaplasia of the cylindrical and plate epitheli as the result of stone formation is causative. The composition of the ne formed plate cells becomes horny, concentric layers form, and, fin nest formation takes place. This, however, is probably true in only a of the cases.

Cancer of the gall-bladder is by no means so rare as was form believed; at all events, primary cancer of the gall-bladder is much frequent than carcinoma of the biliary passages. Approximately, ca is found in females 5 to 7 times more frequently than in males. Whe besides cholelithiasis, the clothing worn by women (lacing) exerts a predisposing influence cannot be determined. Carcinoma of the gall-bladder usually occurs between the 40th and 70th (or possibly the 80th) years of life. In the majority of cases it develops after the age of 50. Frerichs mentions a case of cancer of the gall-bladder in a patient aged 26.

SYMPTOMS

The symptoms, unfortunately, are, in the main, very vague, and the clinical picture of carcinoma of the gall-bladder is by no means typical.

In a certain number of cases, at most in $\frac{1}{6}$, we will learn from the history that cholelithiasis has preceded. But the frequent absence of this proof, naturally, does not disprove the theory that cancer of the gall-bladder has developed from the irritation of stones. As is well known, concrements in the gall-bladder do not always cause subjective and objective diagnostic symptoms, and cases of this kind, particularly, show a tendency to the development of carcinoma. Among the subjective symptoms in 60 per cent. of the cases, pain in the gastric and hepatic regions (gall-bladder) is mentioned, partly paroxysmal, partly of a spasmodic character ("gastric pain"), partly continuous and then less severe. The hepatic region, as a rule, is markedly susceptible to pressure. There is loss of appetite, frequently absence of free hydrochloric acid in the gastric contents, usually constipation, rarely diarrhea. Jaundice of varying degree occurs in about $\frac{2}{3}$ of all cases. Enlargement of the liver develops in consequence of stasis of bile or metastasis. Enlargement of the spleen, particularly in long existing jaundice, is not very rare. Ascites is comparatively frequent; its varying appearance, the jaundice, and the successive parallelism and divergence of biliary and portal vein stasis, I shall consider later. Jaundice is observed more than twice as frequently as ascites. Moderate or even decided fever, invariable emaciation, muscular weakness, cachectic appearance, insomnia, and, with decided jaundice, the hemorrhagic diathesis as well as comatose conditions, are often observed.

Following Courvoisier, the symptoms are designated as "*uncertain ones*"; they might also be called "*inconstant ones.*" In contrast to these, alone or combined with them, tumor of the gall-bladder is a significant sign which makes a diagnosis possible.

Of all the *methods of examination* employed in such cases, palpation is the most important. Percussion usually gives us no reliable data. Relative dulness, at least, can generally be determined over the tumor of the gall-bladder, and, passing upward, this is usually merged in that of the liver. If, however, the intestine covers the elongated, sausage-like gall-bladder between the fundus and the border of the liver, which sometimes occurs, even this relative dulness is absent, or, at least, its connection with liver dulness is uncertain. For palpation, the patient assumes the dorsal decubitus, draws the knees up, and opens the mouth. The head of the patient should be low, the respiration quiet. The seat of tenderness is slowly palpated, and we become convinced that the median region above the navel and the region of the typhlon (cecum) is not conspicuously sensitive. To prevent muscular contractions, we must gently probe with the tips of the fingers into the deeper portions, the fingers being kept extended. Rheinstein has recently called attention to a method of palpating the gall-bladder. The best results are obtained by bimanual examination with the patient in the recumbent posture, and, if necessary, also while the patient is in an erect position. The physician, standing to the right of the patient, places his left hand upon the right lumbar region. The index finger rests upon the twelfth rib, the tips of the fingers being directed toward the vertebral column. The right hand is placed upon the anterior abdominal wall of the patient, the ulnar border being about parallel with the linea alba. The tips of the fingers palpate toward the lower border of the ninth rib, which brings the second finger to a point corresponding with the parasternal line. With the left hand strong pressure is made upon the lumbar region internally and anteriorly, the right hand exerting pressure at the same time so that the liver is simultaneously forced somewhat down ward, and the gall-bladder presses upon the lower half (also presses in wardly) of the right kidney, where it may be palpated. If the patien is standing, the right hand of the physician passes below the anterio border of the liver, so that the four fingers press the fundus of the gal bladder downward, while the thumb exerts pressure anteriorly and the le hand grasps and helps to immobilize the liver. The latter method particularly satisfactory if the gall-bladder be but little or not at a enlarged. In Wijnhoff's method of examining the liver, the patient si upon a chair, flexes the legs, and bends the upper portion of the bo anteriorly. The physician is seated at the right of the patient, and pa pates the anterior region of the liver, grasping it from behind. Palpati of the gall-bladder is, of course, greatly facilitated by anesthesia. Acco ing to Naunyn, if we occasionally fill the stomach with gas, we may convinced that the tumor in question corresponds to the gall-bladder; this case it moves decidedly to the right, sometimes also upward; it also forced more in the direction of the abdominal walls, may be m readily palpated, and even becomes visible. A renal tumor, on the ot hand, disappears when the stomach and intestine are filled with air (wate Exploratory puncture, of which Harley, in particular, made improper u is no longer employed.

In about one-fourth of all the cases, even in the later course of disease, the carcinomatous gall-bladder cannot be palpated. It is t either covered by the enlarged liver, or has become adherent to the stom and small intestine, deep in the abdominal cavity, or is markedly contrac The tumor present is not always actually situated in the gall-bla region; its site varies decidedly (hypochondrium and also epigastrium, right mesogastrium, and rarely it is low down at the level of the crest of the ilium). Taylor believed that the enlarged gall-bladder was always situated close to a line drawn from the normal position of the fundus of the gall-bladder (from the anterior end of the cartilage of the tenth rib) obliquely to the left and downward so that the median line is crossed somewhat below the navel. This "diagnostic" line is of no value. The typical form of a tumor of the gall-bladder is oval, but it may also be cylindrical, and a globular, a sausage, or cucumber form is also observed. The upper border of the tumor is usually difficult to define; sometimes, it is true, we may perceive distinctly that the tumor protrudes below the border of the liver; that is, we may trace it to the right and to the left sharply up to the tumor, and also inward from the periphery. The tumor follows the expiratory excursion, and may be displaced laterally; where there is connective tissue fixation, the mobility is lost. Sometimes only dilatation of the gall-bladder is apparently present, although a carcinoma of the wall (neck of the gall-bladder) may exist. The determination of an uneven, nodular, tough composition of the tumor is significant. Fluctuation, particularly with a "smooth" tumor, is frequently found.

DIAGNOSIS, DURATION, AND PROGNOSIS

The *duration* of the disease, from the onset of positive symptoms until death, is scarcely more than from 6 to 8 months, and in about one-half of all cases is less than 4 months.

It is often difficult or quite impossible to recognize carcinoma of the gall-bladder, even in advanced stages of the disease; but, in the majority of cases, by repeated careful examinations, and a minute weighing of all the conditions present, it may be correctly diagnosticated. The tumor of the gall-bladder is occasionally looked upon as a wandering kidney, a tumor of the omentum, as a corset lobe of the liver, and even as cancer of the pylorus. Such errors are caused by the deviating position, the unusual form, consistency, etc., of the tumor. Of much greater practical importance is the fact that comparatively often operation is performed for a supposed cholelithiasis, and laparotomy discloses a more or less well-advanced cancer. In a differentio-diagnostic respect it is here of the utmost importance that, as well as a hard tumor, we may find no sharply circumscribed tumor of the gall-bladder but only a more diffused enlargement (resistance) of the entire gall-bladder region including the adjacent liver, and thus the growth of the tumor can be recognized. Inversely, sometimes enlargement of the gall-bladder from other causes (the presence of stones or a mere thickening of the walls of the gall-bladder) has been mistaken for a malignant growth. In cases in which a tumor in this region cannot be determined with certainty, the liver may be definitely shown to have undergone carcinomatous degeneration; if no primary carcinomatous disease can be found in other regions of the body, and there is decided pain upon pressure in the region of the gall-bladder, and the patient is a woman, carcinoma of the gall-bladder should be thought of as the underlying affection. If ascites be a complication, splenic enlargement is an important diagnostic factor; if cirrhosis can be excluded, ascites and enlargement of the spleen favor a neoplasm upon the concave surface of the liver. Often the course is calculated to facilitate the recognition of the affection. Examination of the urine and of metabolism does not enable us to form an early conclusion as to the presence of cancer. In carcinoma of the gall-bladder, however, a degree of cachexia soon appears such as is observed in cholelithiasis only late or after permanent occlusion of the common gall-duct. Incarceration from stone may occasionally last for years; a case of carcinoma of the gall-bladder usually terminates in less than six months. If, however, we wait for the time when a hard, oval, nodular tumor appears in an area corresponding to the gall-bladder, and follows the respiratory excursions, and swelling, a hardening, and a nodular consistence of the liver as well as jaundice and ascites have been superadded, the autopsy will then rarely reveal an error in diagnosis, but the time for professional aid will always (or almost always) have passed!

After these statements, the fact that the *prognosis* of carcinoma of th gall-bladder is always exceedingly grave needs no explanation.

TREATMENT

The only *radical treatment* is extirpation of the gall-bladder, and, as
rule, simultaneously more or less extensive resection of the liver. Chole
cystectomy was first performed by Langenbuch in 1880, and since tha
time there have been numerous reports from surgeons who have studie
the indications for this operation. The controversies concerning this poin
do not belong here; but they express the unanimous opinion that extirpa
tion of the gall-bladder for carcinoma, with or without simultaneous r
section of the liver, up to the present time has been absolutely unsuccessfu
In so far as reports are at hand, almost all the cases operated upon su
cumbed to early relapse, to metastases, or other complications (Tait, Czern
Riedel, Bardenheuer, Hochenegg, v. Winiwarter, Socin, Heidenhain, v. M
kulicz, Löbker, and others). Kehr is, therefore, of the opinion that
cancer of the gall-bladder has been determined with certainty (explorato
incision), it is wise to refrain from any attempt at a radical cure. Cy
tostomy may, under such circumstances, bring about transitory ameliorati
of the condition; with drainage of the gall-bladder the pain ceases; bu
on the other hand, in consequence of the cancer attacking the cystic du
a distressing mucous fistula develops. Löbker attempted in 4 cases
relieve the pain caused by a simultaneously present empyema of the ca

cinomatous gall-bladder by performing cholecystostomy with a possible removal of the stones. All of these patients died soon afterward, two of them directly in consequence of the operation, and sooner than would otherwise have been the case.

If, therefore, we wait for the time when the diagnosis can be positively made, a successful radical therapy is impossible, because the disease is no longer confined to the gall-bladder alone, or to its immediate vicinity, but has extended to neighboring organs, where it has led to the formation of metastases, to perforation, etc. Heddaeus, therefore, proposes that we regard the symptoms designated by Courvoisier as " uncertain ones " as indications for exploratory laparotomy, and that in the given case we make an exploratory incision into the gall-bladder to determine the condition of the mucous membrane. Thus he would attack the evil at its root, would arrest the development of new formations, and thus avoid the danger of too extensive an operation, which inevitably and rapidly leads to death. As in many cases, we refer the onset of the carcinoma to the fundus, he hopes in this way, even by a partial resection of the gall-bladder, to arrest the further advance of the carcinoma. In all cases in which the neoplasm originates from the cystic duct or from the neck of the gall-bladder, this later method would, *a priori,* be excluded, but the resection of an implicated portion of the liver would not cause insurmountable difficulty. The question whether it is justifiable in cholelithiasis, and under certain circumstances, to undertake an exploratory incision may be answered by every physician in the affirmative, as the danger of this operation is in no sense commensurate with the ultimate advantage gained or with the damage which might ensue from neglecting it. Exploratory laparotomy is justifiable when we have long-continued and severe pain, when the patient is unable to follow his occupation, when there is loss of strength and decided emaciation, when internal treatment is futile, when there are negative or entirely obscure findings in the liver and gall-bladder, and those symptoms are excluded which make occlusion of a common gall-duct by a tumor very likely. Patient and physician will be much less willing in the frequent cases in which carcinoma has led to the development of a determinable tumor with but slight subjective difficulties. I doubt, therefore, that the proposition of Heddaeus will largely reduce the number of persons succumbing to cancer of the gall-bladder.

The difficulties which attend a late operation for carcinoma of the gall-bladder may be met by considering all of the practical consequences of the previously described etiology of cancer, and by directing the treatment chiefly to the removal of the cholelithiasis. No practitioner, at the present time, can escape the conviction that in numerous types of cholelithiasis in regard to which physicians and surgeons, fortunately, have recently more nearly approached unanimity of opinion, the surgeons are the ones most likely to afford relief, and are, therefore, the proper therapeutists. The operative method which is accompanied by comparatively slight immedi danger to the patient, which demands the sacrifice of an organ not ab lutely necessary to life, and which is by far the most important seat gall-stone disease, which removes the first germs of carcinomatous form tion in the wall of the bladder irritated by the stone, is *cholecystectom* Performed at the proper time, not only for the threatening carcinoma b also for general *curative* and prophylactic purposes, an extension of t indications for extirpating the gall-bladder in the cases in which cho lithiasis is limited to this organ is desirable. At all events, even chro inflammatory conditions of the gall-bladder with a thickened wall a those anatomical changes of the epithelium which may be looked upon precursors of carcinoma, should be included in these indications. Nat rally these points must be left for the surgeons to decide; it seems to that in this respect their opinions are at present undergoing a change. T cases in which Löbker undertook to perform cholecystectomy on accou of supposed empyema, while tissue suspiciously resembling tumor was noted by the operator, and in which Grawitz showed microscopically beginning carcinoma, should increase the number of advocates of ea cholecystectomy more than it has. Whether we shall in this way succe in diminishing the percentage of patients with gall-stone disease w acquire carcinoma of the gall-bladder, future experience will alone deci One point must certainly be touched upon. It has been explained ab that in those cases in which stones remain encysted in the gall-blad for years, there is a tendency to the development of cancer of the g bladder producing actual spasmodic paroxysms without inflammatory pr esses. The law still operative with many surgeons: "Quiescent gall-sto are not subjects for treatment," must therefore be construed with so limitations. Enlarged gall-bladders tensely filled with stones, particul where there is a hereditary predisposition of the patient to cancer, sho be extirpated on principle. The question is simply this: How does physician observe this except quite incidentally, and how are we to tain the patient's consent to an operation if we tell him that, in propor to cholecystitis which is very frequent, cancer is a very rare disease?

2. OBSTRUCTIONS OF THE COMMON GALL-DUCT BY TUM

CLINICAL FORMS

In clinical study it is difficult to separate the consideration of *carci of the biliary passages external to the liver* from that of obstructio the common bile-duct and of the hepatic duct in general.

Occlusion of the ductus choledochus may result primarily fro *anatomical, abnormal, duodenal insertion and twisting* of this passa its entrance into the intestine, whereby, in exceedingly rare cases, ev youthful individuals, a valve-like closure and consequent extreme cystic dilatation occurs (Konitzky). This colossal dilatation of the gall-duct may then form a resistant tumor in the right half of the abdomen, and the bulging wall of the passage in itself causes an increasing compression from without upon the end of the gall-duct extending into the intestinal wall. In a child aged 2 months, with decided jaundice, an exploratory laparotomy was performed (by Witzel), and the subsequent autopsy showed a *congenital marked narrowing of the cystic and common ducts, as well as the absence of the hepatic duct. Congenital obliteration* of the ductus choledochus, probably as the result of *fetal syphilis,* is somewhat more frequent. Soon after birth such children are decidedly jaundiced without fever and rapidly perish. This has sometimes repeatedly occurred in the same family (Donop, Binz, Glaister, Schüppel).

Chronic occlusion of the ductus choledochus by stone (*incarceration of calculi*) and the infrequent *cicatricial obliteration* of the same in consequence of cholelithiasis are of far greater practical importance. The very rare *adhesions* due to *propagated* (?) *inflammations* from the intestine in enteric fever (Lebert), in enteritis (Musser), in the puerperium, chronic peritonitis (Andral), and in benign pyloric stenosis (Richard) at least deserve brief mention here. The greatest interest attaches to *neoplasms of the common bile-duct.* We must also mention occlusion by *foreign bodies other than stones,* and by parasites (echinococcus cysts, ascarides, and varieties of distoma). *Hydatid cysts* always develop in the liver, from which they penetrate to the larger biliary passages. Years ago a patient of mine was operated upon by v. Eiselsberg for echinococcus of the liver. On account of extreme chronic jaundice which was simultaneously present I diagnosticated a closure of the common gall-duct by daughter-cysts; hepatic echinococcus cysts and a cholesterin stone were found in the gall-duct and were at the same time removed. Besides occlusion from stones the hepatic duct is sometimes occluded by *callous cholangitis* (rare), by *neoplasms,* by *fetal syphilis* and by *parasites.*

In addition to the obstructions to the permeability of the large biliary passages which have been mentioned, there are numerous "vinculo quasi injecto" which *compress* the lumen (or simultaneously compress and infiltrate it). These are either actual neoplasms, or tumors in the widest sense of the word, and connective tissue proliferations. These obstructions may originate in the *porta hepatis*: *hydatids, aneurysms* of the hepatic artery (Niewerth), of the abdominal aorta (Stokes), of the superior mesenteric artery (Wilson, Gairdner), enlarged *tuberculous, carcinomatous, lymphomatous, amyloid* glands of the porta hepatis, along the hepato-duodenal ligament, and near the mouth of the common gall-duct, circumscribed adhesive perihepatitis at the hilus in affections of the liver, *syphilis ad basin, ulcers of the stomach and duodenum, mediastinitis* after carcinoma of the breast (Smolenski), and carcinomata. Besides compression, cicatrices in the region of the porta hepatis also cause torsion of the common gall-duct, so that it is bent and follows an irregular course. We must also consider changes in the size of the *pancreas* (cystic dilatation of the duct of Wirsung (Wyss and others), *suppurative pancreatitis* (Frison), sclerosis (Déjerine), principally, however, *carcinoma of the head of the organ,* and, finally, *malignant disease* and *cicatricial stricture of the duodenum.* Primary *carcinoma of the gall-bladder* may by continuity creep through the cystic duct to the common duct and the hepatic duct, and, besides producing angiocholitis, may cause compression and stenosis. Occasionally the enlarged tensely filled gall-bladder (in consequence of occlusion of the cystic duct by stones), or dropsy (empyema) of the gall-bladder without concrements, may compress the common duct (Ramskill Socin, Kümmel). Stones embedded in the cystic duct may also occlud the common duct (Wilson, Bardenheuer). Even the relaxed gall-bladde whose tonus is impaired by long-continued distention due to inspissate bile, or by a condition of fulness of the common gall-duct, may cause com pression leading to biliary stasis (Petit, Jacob and Cyr). Occasionall such a gall-bladder may be expressed. Adhesions of the gall-bladder t the duodenum and colon may cause a volvulus of the ductus choledochu and the duodenum, in particular, is drawn to the right and upward. Su *twisted stenoses* are often increased by the simultaneous presence of ston It is also possible that a *wandering kidney* by its tense ligaments m cause a volvulus of the common bile-duct, a possibility which Weisker nies, but which, nevertheless, exists. On the other hand, tumors of the rig kidney may cause obstruction (Schüppel). Even tumors of the colon, t omentum, the retroperitoneum, and of the ovary and uterus, must be c sidered as possible obstructions to the flow of bile.

If we discuss only the cases in which occlusion of the common g duct develops an actual, extensive (absolute), biliary stasis, we find an rysms and enlargement of the glands of the porta hepatis, as well as c of the pancreas, comparatively rare. *Perihepatitic* and *pancreatic ind tions* are, as Courvoisier states, more important. Of most serious im in this connection are *concrements and neoplasms in the ductus ch dochus, carcinoma of the head of the pancreas and of the duodenu* the region of the plica Vaterii. *Secondary malignant infiltrations* of biliary passages by *carcinoma of the gall-bladder* must also be included The most significant causes of obstruction of the choledochus are also most frequent, and, therefore, of the greatest clinical interest. The d choledochus in its passage to the duodenum burrows for itself a furro the head of the pancreas, or it perforates the same, and unites with ductus Wirsung near its mouth. The upper horizontal portion of the denum, as well as the two other portions, are reached from the he the pancreas with which the two duodenal areas closely unite and a bow-shaped intestinal portion. The point where the common gall enters the duodenum is the left concave surface of the pars descendens duodeni. If the ductus choledochus does not penetrate the head of the pancreas it passes behind it, and enters the substance of the pancreas just before its junction with the pancreatic duct. The two passages run alongside each other for a short distance, then perforate obliquely into the longitudinal musculature, continuing their course for some time below the mucosa, and finally, by a common orifice, pass into the diverticulum of Vater. Sometimes, however, they terminate separately. Compression is most likely to occur when the ductus choledochus passes through the head of the pancreas; but these topical relations explain why a tumor which has developed in the ductus choledochus furnishes a picture so nearly analogous to that of a tumor at the head of the pancreas, or of a carcinoma, or even of a cicatricial stricture in the region of the diverticulum Vateri duodeni. From an anatomical standpoint the question has arisen (Pic) whether carcinoma of the diverticulum of Vater should not be referred to the pancreas rather than to the duodenum. No matter what the conditions are, the obstructions which obliterate or compress the ductus choledochus must also mechanically implicate the pancreatic passage, which is not infrequently subject to dilatation (cystic).

In practice the question usually resolves itself as follows: Jaundice (stasis) reaches its acme of development either rapidly or gradually; if it does not disappear after a short time, it merges into a chronic icterus. In prolonged "*catarrhal*" *jaundice* (lasting 2, 5 or even 9 months), when the symptoms of gastrointestinal catarrh have subsided and the signs of pure, chronic, biliary stasis have become prominent, particularly a disturbance of the general condition, with weakness and emaciation, the clinical picture of biliary occlusion from other causes (incarceration of stone compressing or occluding neoplasms) may resemble it so closely that the diagnosis is made clear only by the further course of the affection (final, complete and permanent restitution of permeability of the biliary passages). Certain diagnostic support will be found in the fact that the onset of the disease, according to our own observation, or after obtaining a history of the cause and clinical behavior, coincides fully with that of catarrhal jaundice; that the occlusion of the biliary passages is incomplete, having been only partial for a time, or even continuously; and that, finally, the liver and spleen have permanently presented only a sign of biliary stasis. But in catarrhal jaundice the hemorrhagic diathesis may also develop! Neusser and Toelg report the case of a man, aged 39, who was attacked by jaundice after partaking of sausages, and who succumbed in the eighth week of the disease after hemorrhage from the urinary passages. The autopsy showed catarrhal cholangitis, with complete impermeability of the ductus choledochus. The hemorrhages began during the seventh week. Nowadays, under similar circumstances (probably, however, before the appearance of the cholemic hemorrhages) an operation (cholecystotomy) years). As a rule, this cirrhosis is without decisive importance for the *decursus morbi.*

If, however, in consequence of any occlusion of the common bile-duct, marked, high-graded icterus stasis develops, there are also quite isolated, paradoxical cases in which this condition does not follow. Such a case, for example, was observed by Hanot and Gombault. In consequence of "chronic gastritis with submucous sclerosis and callous retroperitonitis," cirrhosis of the hilus of the liver had formed and completely obstructed the ductus choledochus, but there was no jaundice, and merely partial occlusion of the hepatic artery and portal vein. Hanot and Gombault assumed that the succeeding disturbance of circulation had limited the quantity of bile formation and, synchronous with the hindrance by occlusion, absorptior was suspended so long as even the smallest quantity of bile passed into th intestine; later the secretion ceased entirely, so that even complete occlu sion of the common bile-duct did not produce jaundice. From a deficienc in formation products, the secretion of bile ceased, and this caused th death of the patient. In this case, the important factor was the simultan ous strangulation of the hepatic vessels. However, I believe it more likel in such exceptional cases that an auxiliary system of biliary canals, whic is also present under normal conditions and serves the purposes of ana tomosis, intervenes and permits the vicarious emptying of an auxilia branch of the ductus choledochus and the like.

Among the previously mentioned pathological processes which wi comparative frequency cause obstruction of the choledochus the followi must be mentioned: *Chronic impaction by stone, carcinomata of the co mon bile-duct, of the head of the pancreas and duodenum, as well cicatricial stricture in the region of the diverticulum of Vater, circu scribed perihepatitis at the hilus of the liver, and, possibly, also enlar ment of the periportal lymph nodes.* In so far as a gastric cancer, o primary carcinoma of the gall-bladder, attacks the ductus choledochus a the hepatic duct, the resulting stasis of bile must be ascribed to the tun of the choledochus provided a fundamental diagnosis cannot be made. is of the utmost importance for the physician to know with certai that an occlusion of the biliary passages by stone or some other ca (tumor) has occurred. If we feel assured that a stone is present in common duct, and in case intermittent fever sets in soon after the colic which is accompanied by jaundice, choledochotomy is to be formed. In chronic obstructive jaundice due to carcinoma, much les to be hoped for from operative interference; even exploratory laparoto according to experience, is badly borne! Causes purely mechanical favor the conception of these two groups of cases: *Calcareous occlu and occlusion of the biliary passages from other causes.* For, as wil shown in the following, a series of other factors may be recognize which, aside from numerous, not rarely insurmountable, difficultie individual observations, the differentiation between chronic occlusion of the ductus choledochus by calculus and tumor may be indicated; as a rule it is almost impossible to distinguish the different cases of occlusion not due to stones. The course is most favorable, as was first indicated by Courvoisier, Leube, and others, and corroborated by Neusser in the diagnosis of *obstruction of the choledochus from pressure in the porta hepatis.* Probably in most of the cases belonging to this group, but by no means exclusively, there is a determinable, characteristic factor, that is, the parallelism between biliary stasis and portal vein stasis evidently caused by the anatomical situation referred to. As symptomatic expressions of the latter, besides ascites and enlargement of the spleen, we must consider diarrhea, gastric and intestinal hemorrhages, and a visible collateral circulation. An intense and almost simultaneous stasis in the portal vein and biliary system (after excluding ordinary hepatic cirrhosis) would lead us to think of enlarged glands at the hilus of the liver and circumscribed perihepatitis *ad basin.* Apart from carcinoma and syphilis, the clinical interest of these processes is comparatively slight. Naturally there are tumors also upon the under surface of the liver, as, for instance, in the case of Quincke, in which a large fibroma was present, but which, on account of its position, merely occluded the choledochus without causing ascites. On the other hand, in the much more frequent cases in which the greatest pressure is on the duodenum, this is a variable condition. Usually jaundice is present for a long time before ascites appears, or the latter symptom does not occur at all (it is most readily produced in carcinoma of the ductus choledochus and of the duodenum itself), that is, during the entire course of the disease it is very slight. Whether the peritoneal effusions of the tumors which we have an opportunity of observing, and which are due to the very small tumors of the intestinal portion of the biliary passages, actually depend upon purely mechanical obstruction of the circulation in the portal vein, I believe to be very questionable. In carcinoma of the head of the pancreas and the upper end of the duodenum, as well as in cancer of the gall-bladder which extends in the direction of the porta hepatis, at least occasionally and in certain stages of the course, the ductus choledochus and the portal vein sustain almost equal pressure.

As is shown by the statistics of surgeons up to the most recent times, in tumors which occlude the distal portion of the choledochus, a positive, diagnostic recognition in the individual case prior to the operation (that is, during the life of the patient) was frequently impossible; if, indeed, a correct opinion with a special localization of the tumor could be given at any time. On the contrary, the operation was usually performed because, diagnostically, the disease was supposed to be present, but, at the same time it was often erroneously presumed to be a case of occlusion of the biliary passages by stones. I shall, therefore, utilize a somewhat extensive and original experience in regard to obstruction of the choledochus by tumor to criticize the differential diagnostic factors which have up to the present time been proposed. Since 1887 I have collected 24 autopsy reports of occlusion of the choledochus not due to stone (in Prague, Vienna, Graz). In another case, metastases due to sarcoma were present, which, starting from a large, visible and palpable tumor of the left ilium, compressed the biliary passage. Twenty-two cases were due to obstruction from carcinoma, the two other cases were attributed respectively to an echinococcus cyst upon the concave surface of the liver and to a cicatricial stenosis of the duodenum after rupture from a stone. In 13 cases we were dealing with cancer of the biliary passages, in 7 with carcinoma of the head of the pancreas, and in 2 with a duodenal tumor. To this must be added 2 cases (with section) of tumor of the pancreas without jaundice, and 3 others, not confirmed by autopsy, of occlusion of the ductus choledochus, in which the diagnosis of carcinoma was made with at least great probability and which, in certain respects, may here be utilized. In contrast with these reports of occlusion of the choledochus not due to stone are 11 original cases in which, by autopsy or operation, incarceration due to stone, to cicatricial stricture of the biliary passages following cholelithiasis or abscess, but without a complicating carcinoma, could be determined. From the literature at hand it is diffi cult to form a conclusive opinion as to the frequency of chronic occlusion by stone compared with that due to pressure from malignant tumors Hilton Fagge, after an experience extending over years, states that per manent biliary stasis with all of its sequelæ occurs twice as often i consequence of malignant disease causing pressure as in pressure fro stones. Murchison knew of only two cases of fatal, uncomplicated, chron jaundice due to stone, and Glover believes every case of this kind wor publishing on account of its great rarity. Robson cautions us in gener against operations for obstructive jaundice, since, as a rule, carcinoma the biliary passages is found. Fiedler found this condition only tw among 800 to 900 autopsies in cases of gall-stone disease (that is, amo 10,000 general mortalities). In 343 cases of cholelithiasis (among 4,3 autopsies) Schloth (in Erlangen) found a stone in the ductus choledoch 9 times, therefore, in 0.2 per cent. of all autopsies, and in 2.6 per ce of all the cases of gall-stones. According to Courvoisier, the proporti are even greater in Basle: In 2,520 autopsies and 255 cases of cholelith sis, stone was found 10 times in the choledochus, i. e., in 0.4 per cent. all autopsies and 3.9 per cent. of all gall-stone cases. The rarity of cases coming to autopsy does not certainly prove the rarity of the co tion; occlusion due to stone need not, as such, be absolutely fatal, for a large number of cases, the calculus eventually finds its way out. 6 non-operative cases collected by W. Braun (Pringle, White, Coper Michaux, Riedel, Thornton) the outcome was fatal in 5, and in the pa of the last named author a spontaneous passage of the stone occurred was followed by cure. Important complications may set in. That chronic jaundice due to a calculus permanently incarcerated in the ductus choledochus is not *absolutely* rare is proven, in the first place, by the experiences of surgeons who are specialists in this line. Nevertheless, according to my own experience, if the physician does not observe "selected" cases, even allowing due weight for the intermissions in the jaundice, he must be familiar with the fact that the severest (fatal) cases of chronic biliary stasis are due far more frequently to obstruction of the choledochus which is not due to calculi. In this opinion Schüppel coincides.

PATHOLOGY

In regard to the special pathology of *adhesive perihepatitis in the porta hepatis* and in *affections of the periportal lymph nodes,* I shall not at this point enter into details. In syphilitic hepatitis marked icterus from compression of the biliary passages is much rarer than ascites. Carcinomatous metastases of the portal glands are peculiar to primary and secondary cancer of the liver, and cause decided swelling. Primary cancer of the gall-bladder leads to the development of cancer in the glands of the hilus and in the porta hepatis, by which the outflow of bile is impeded and the portal vein is compressed. Ascites is found here much more frequently than enlargement of the spleen. Intestinal hemorrhage, even dilatation of the collateral venous trunks, is observed in this condition. It must be borne in mind that the portal vein may also be occluded and ascites result from complicating cirrhotic changes (carcinomatous cirrhosis) or from obstruction (compression) of the veins of the liver. In leukemic and lymphomatous tumors, the portal lymph-glands are also frequently enlarged. But if the liver is not correspondingly changed from other causes (for example, in certain cases of pseudo-leukemia with chronic relapsing fever) decided jaundice is very rarely present, and ascites usually occurs late. (Chronic peritonitis of leukemia belongs to another category.) In the diagnosis of these conditions, therefore, aside from the relation between the biliary and portal vein stasis, it is necessary, above all, that we find in some part of the body the primary organic affection which would produce secondary carcinomatous disease of the glands of the hilus, or the proof of an analogous affection of other peripheral lymph-glands (leukemia, pseudo-leukemia), or that we discover signs of constitutional syphilis, etc.

The case mentioned above of *compression of the biliary passages* upon the lower concave surface of the liver by an *echinococcus cyst* may be here briefly quoted.

A woman, aged 30, wife of a meat-chopper (butcher), who had previously suffered from enlargement of the liver and ascites, and afterward from gradually increasing jaundice, subsequently noted a more rapid and more decided enlargement of the abdomen with slight swelling of the feet. Severe abdominal pains were added.

On June 8th, 1888, she was visibly emaciated, markedly jaundiced, her color brownish-gray, her temperature between 100° F. and 102.9° F. Upon the right side of the neck a mass of partly isolated, partly connected, lymph-glands of the size of a small apple; in the left supraclavicular fossa isolated intumescent lymph-glands from the size of a millet seed to the size of a bean. Left-sided pleurisy. Over both lungs piping râles, occasionally crepitation. Free fluid in the peritoneal sac. The liver enlarged, of coarse consistence, its surface smooth; the border, which may be readily palpated, is dull. Gall-bladder not palpable. On percussion, at least, enlargement of the spleen. Urine very dark, yellowish, foamy; chlorids in normal amount.

Pain in the right hypochondrium continues. The temperature rises each evening above 102.2° F., early in the morning it falls below 100.4° F. Feces not entirely devoid of bile, very rich in fat. No bacilli in the expectoration. June 18th: Pericarditis. June 19th: Complete absence of bile from the feces. Hemorrhagic diathesis, hemorrhages into the skin, from the nose, hemorrhage of the gums. June 23rd: Marked loss in strength. June 25th: Subnormal temperature. June 26th: Death.

Autopsy: Echinococcus cysticus multiplex cum compressione ductus hepatici et cystici, subsequente ictero gravi.—Tuberculosis chron. gld. lymphaticar. colli. Pericarditis, pleuritis sin. tuberculosa cum exsudato fibrinoso-haemorrhagico. Peritonitis tub. The liver appeared enlarged to 1½ times its normal size; upon the upper border of the right lobe between the gall-bladder and the hepato-umbilical ligament an uneven tumor of the size of a man's fist was noted, and at the vault of the liver, under the capsule, a whitish tumor the size of a hen's egg was found. The first mentioned tumor bulged decidedly downward, so that around it the hepatic duct and the beginning portion of the common duct were found empty and pressed flat. Owing to this dragging, previously mentioned, it was difficult for the sound to pass the biliary passages. The cystic duct also encircled the lower border o the tumor, and this, too, markedly dragged. The walls of the distended biliar passages were decidedly atrophic. The gall-bladder contained but little bile. A thir tumor of the same character was found deeply embedded in the right lobe of th liver; the liver tissue showed an increased consistence. A brownish-green icteroi condition was observed. The distended intrahepatic biliary passages contained muc mucoid, watery, yellow bile. The enlargement of the spleen was very considerabl The tumor presented the characteristic features of echinococcus cysts.

As is well known, compression of the large biliary passages upon t concave surface of the liver combined with jaundice in consequence the stasis of bile is rare, particularly in echinococcus cysts of the live according to Finsen it occurred only 7 times in 167 cases, according Neisser 20 times in 388 cases. An analogous effect upon the portal ve or vena cava is infrequent. Damaschino once found a large echinococc sac in the right lobe of the liver, pressing upon the ductus choledoch in the porta hepatis; in this case another small hydatid lay along t longitudinal fold of the bile ducts. In my case the complete closure, t icterus gravis, the hemorrhagic diathesis, the hepatargy, as well as preceding ascites (apparently the peritonitis occurred late), were m remarkable.

I have seen no case in which *enlargement of the pancreas other t* *from carcinoma* could give rise to complete occlusion of the common duct. Evidently obstructions from cystic dilatation of the duct of Wirsung (Wyss) from purulent pancreatitis, and even from sclerosis, are rarities. On the other hand, tumors of the head of the pancreas with actual tumors of the choledochus may in their frequency enter into competition (in this relation cancer is first, lymphosarcoma is rare). Many authors regard cancer of the pancreas as even the usual cause of obstruction of the common bile duct. In my own observations, as has been mentioned, there are 13 cases of cancer of the choledochus (among these 8 positive primary neoplasms), and, in contrast, 7 of primary carcinoma of the head of the pancreas. No doubt primary cancer of the duodenum beginning at the diverticulum of Vater is much more uncommon. One of my patients with cancer of the pancreas was only 25 years old, the age of the others varied between 47 and 70. In regard to sex my experience was uniform with that of all other observers, in that among my cases there were but two women. Of the etiology of carcinoma of the pancreas, we know as little as of the cause of cancer in general. Anatomically, the epithelioma starting from the epithelium of the glands as well as from the cells of the ducts usually presents itself as scirrhus, more rarely as a medullary tumor, and most rarely as a colloid cancer. Most often (in over 60 per cent. of all cases) the seat is in the head, and the tail of the gland is usually exempt. The growing neoplasm of the pancreas much more frequently proliferates to the duodenum (also to the stomach) than the duodenal (pyloric) cancer to the pancreas (in spite of Olivier's conclusions). The head of the pancreas attacked by cancer is often decidedly enlarged, up to the size of a child's fist or larger; during the life of the patient, however, even to the end, the tumor is not palpable (this was true of 6 out of 7 of my cases), which is in contrast to the view of Leichtenstern, who maintained that it was very frequently palpable. Sometimes the pancreatic duct is occluded, and if the resisting tissue still secretes fluid, stasis of the secretion with cyst-like dilatation of the duct occurs (Bolds). As may be readily surmised from the previous description, the ductus choledochus with very rare exceptions meets the same fate, but the mode of implication of the biliary passage varies. If this reaches the intestine without entering the substance of the pancreas itself, under some circumstances compression alone occurs. The common bile duct remaining free may, however, be actually surrounded or infiltrated by the carcinoma. If it permeate the head of the pancreas, a malignant growth develops in its walls, perforates it, and occludes the lumen. Under these varying conditions decided stenosis of different grades is found rather than absolute closures. Dilatation of the biliary passages is the rule, dilatation of the gall-bladder is found in at least 70 per cent. of all cases that have been observed, in all of my own cases with the exception of one (Clässen, Arcelet, Krieger, Legendre, Rotch, Lachmann, Wyss, Frerichs, Murchison). Besides cancer of the pancreas, I saw one case of chole(cysto)lithiasis.

The large blood-vessels in the vicinity and the solar plexus are also implicated in cancer of the pancreas, and not rarely, as is the case with the duodenum; in my 7 cases perforation of its wall was found 3 times. Even the ureters may be compressed; this is partly due to pressure, partly to continued carcinomatous growths, and, in some measure, to the development of tough, strand-like thickenings, with volvulus of portions of the intestine, etc. Apart from the mesenteric lymph-glands and the peritoneum, extensive metastases in distant organs are said to be rare; I found several quite isolated ones and even general carcinosis. In three instances I saw numerous secondary cancerous nodules in the liver. At the autopsy of one of my cases a tough blood clot was found which had been lodged behind the stomach and correspondingly presented the shape of a half-filled stomach. The blood originated from the malignant pancreas, upon the posterior wall of which and adjoining the blood focus, two lacerations were found, more than 3 cm. in length and running a zig-zag course through the serosa and the sub-serous connective tissue.

At least briefly, both observations of *carcinoma of the pancreas* are to be mentioned in which the ductus choledochus *remained free.*

The *first* of these cases occurred in a cheese-maker, aged 59, who was repeatedly the subject of trauma of the abdomen. Upon muscular exertion the patient suffered from pain of a neuralgic character in the upper abdominal region, deeply localized between the pit of the stomach and the navel, and radiating toward the back. For four months he had severe diarrhea, and there was great emaciation (loss of 13 kilograms). Upon the 5th of November, 1898, there was no fever, no jaundice no palpable abdominal tumor, no ascites. Liver enlarged and permeated wit nodules. Gall-bladder not palpable. Spleen of normal size. Except in the inguina region, no enlarged glands. No edema. Four hemorrhagic stools daily.

By the administration of two test-meals the amount of free HCl in the gastri contents was proven. The stomach was not dilated. The diarrhea was somewha improved (milk diet). No butter stools; no marked alimentary glycosuria. Up t the time of death (November 19th) a further loss in weight of 5 kilograms.

Autopsy: Carcinoma of the body of the pancreas, the head remaining free. Begi ning rupture into the stomach. Hepatic metastases.

The *second* case was that of a locksmith's apprentice, aged 18, who, after qui prolonged constipation, was attacked toward the end of May, 1900, by celiac ne ralgia and vomiting. The patient observed a rapidly growing tumor in the upp abdominal region; some portions of the feces (after purgatives) were said to "white like bacon." Upon the 7th of June, the patient was still well nourished a without jaundice. There was no fever, no edema. Bilaterally upon the neck a also in the right axilla small course lymph-glands were palpable. A little to right of the epigastrium, between the median line and the arch of the rib, was prominence about 8 cm. in diameter, coarse, globular, but nowhere fluctuating, insensible to pressure. The tumor was deeply adherent, situated in front of abdominal aorta behind the stomach, readily displaced laterally, and also somew movable upon change of position and with inspiratory excursions. Liver and spl not enlarged. The colon which, on account of inflation with gas, could be rea seen and palpated, surrounded the tumor from below, and in front the stomach peared to be forced upward. The latter organ was not dilated.

Constipation continues. The stools are stained with bile; they contain numerous unchanged muscular fibers and starch granules, and are apparently rich also in fat. (Fat-splitting not investigated.) The urine contains no indican. The gastric contents (2 test-meals) show absence of free HCl; the total acidity varies between 1.1 to 2.1 to the thousand HCl. As the filtered gastric contents have a brownish appearance, the absence of free acid is probably due only to regurgitated bile. Decided alimentary glycosuria cannot be proven. The tumor grows rapidly. On account of the torturing attacks of neuralgia coeliaca, and at the urgent entreaty of the patient, exploratory laparotomy was performed (Prof. Nicoladoni). This demonstrated that the duodenum had been considerably narrowed by the tumor. Death upon June 28th.

Autopsy: The tumor to the left reaches to the duodeno-jejunal flexure which appears to be forced to the left and upward. The duodenum also appears to be displaced to the right. The permeable ductus choledochus runs its course upon the right surface of the tumor. The right portion of the pancreas is granular, tough, and shows a whitish discoloration; the tumor extends across this portion of the head, and includes a portion of the retained tail of the gland. Two-thirds of the pancreas has been obliterated by the neoplasm, the duct of Wirsung is markedly dilated. In the vena lienalis is a non-obstructing thrombus from the tumor.

I believe it is not too much to declare in these two cases, in the first on account of the typical pains, the diarrhea and the normal gastric function, in the second from the topical relations of the tumor and the fact that biliary stasis did not occur, that a probable diagnosis is possible. Furthermore, in both cases the reason for the absence of obstruction of the choledochus is quite obvious; the carcinoma did not develop from the head of the pancreas. No explicit and unanimous reports are found in literature to show how frequently persistent impermeability of the duct occurs in cancer of the pancreas involving the body of the gland, but the most reliable reports are furnished by Wyss.

Similarly as in the vessels, a carcinoma of the liver may directly implicate the *biliary passages*, forming a tumor mass extending into the large biliary passages and the intrahepatic ducts, and leading to occlusion of the same, thence to biliary stasis. Benign neoplasms arising from the ductus choledochus and the hepatic duct were early mentioned in literature (the reports of Courvoisier are not complete, Alexander Benedictus, 1508, Marcellus Donatus, 1588). Valuable descriptions have been published since 1840 (Durand-Fardel, W. Stokes, van der Bye). Among the forms described were "fibroids" (Albers), "fatty tumors," i. e., presumably lipomatous tumors (Wardell, Bouisson), fibrosarcomata (metastatic, Schüppel), adenomata, myoadenomata (Calzavara), cystoadenomata (Hippel). As in the bile organs in general, here also *carcinomata* are far the most important tumors, and the ones most frequently found. In a number of cases, it was microscopically proven that they originated in the glands of the bile ducts. The large bile ducts, particularly the central portions, are well supplied with glands. These are found in profusion in the hepatic duct, are less plentiful in the upper portion of the ductus choledochus, and in its lower portion least numerous. The body of the gland, constructed of acini, lies like the crown of a stone pine in the connective tissue walls (Riess). A mucoid secretion is ascribed to it, but it is still doubtful whether it contains true mucin. After enlargement of the gland vesicles, perhaps by coalescence, the cylindrical epithelium proliferates markedly, and permeates the lumen in the form of tubes. Connected with this is a proliferation into the stroma, and the gland vesicles are transformed into microscopically small, cancerous nodules (M. Howald). Usually the stroma is profuse, and the cancer shows the characteristics of scirrhus (Dieckmann). In rare cases the conditions are those of colloid cancer. The neoplasm first filtrates the wall of the biliary passage which it narrows and later occludes. The mucous membrane above the infiltrate may for a long time remain wholly or partially intact. The neoplasm assumes the shape either of a ring or a polypus; occasionally its formation resembles that of villi. Throughout the course the tumors are almost always of small size. Often their diameter does not amount to more than 1 cm., and in length they are comparatively short; some appear to attack the wall only to an extent of 5 cm. Nevertheless, tumors from the size of a walnut to that of a fist have been observed. Their concealed position and small size almost invariably make palpation of tumors of the choledochus impossible. In accordance with the well-known laws of localization for cancer in general, carcinomata of the bile passages are found chiefly in the most narrowed areas of the canal (at the mouth of the common bile duct, at the confluence of the cystic and hepatic duct, at th point of bifurcation of the hepatic duct). At the autopsy of my cases 7 times a tumor was found in the vicinity of the diverticulum of Vater or, at least, below the middle of the course of the common duct, whic was about 8 cm. in length; in 3 cases carcinoma near the junction of th hepatic duct and the cystic, and once only in the hepatic duct. In a other case, there was a contact carcinoma of the ductus choledochus co joined to a primary tumor of the gall-bladder, in another a projecti cancer of the cardia ventriculi attached to the gastrohepatic ligament, a another of the common bile duct and cystic duct. In passing it may remarked that metastatic cancerous nodules are also found isolated in t ductus choledochus. Clinically it is of little significance whether the c cinoma be primary or secondary, since the prognosis of a cancer of t gall-bladder, for example, one which by proliferating occludes the cho dochus, is in any case still more serious than that of a primary tu in the bile duct. The exact localization of the origin of the neoplasm, may be readily understood, sometimes is difficult. In two of the se cases of carcinoma of the ductus choledochus which have been refer to, the lowest portion, corresponding to the diverticulum of Vater, tured into the duodenum; in one there was simultaneous proliferatio the pancreas. Inversely, primary carcinoma of the duodenum may only include the mouth of the choledochus, but, invading deeply, may also occlude the bile duct. The ductus choledochus, below the insertion of the cystic duct, may also be occluded by the tumor mass which burrows through the latter to the gall-bladder, where, in the narrow portion which is immediately connected with the cystic duct, it may be of very small size. That the neoplasms actually originate in these small masses may be readily recognized from the fact that the secondary growth permeates the entire wall of the gall-bladder and also attacks the substance of the liver in its immediate vicinity. I have never seen a neoplasm of the cystic duct which was not connected with carcinoma of the gall-bladder or of the ductus choledochus (hepaticus). According to some statistics (Courvoisier), in 19 cases of cancer of the choledochus the origin of the neoplasm was 9 times in the beginning of the duct near the junction of the cystic and hepatic ducts, 3 times in the middle, 7 times in the pars intestinalis, that is, in the ostium. Carcinomata of the gall-ducts are evidently rarer than those of the gall-bladder. Among the causes of obstruction of the choledochus by tumors, carcinoma of the pancreas, from its frequency, can alone be considered. Etiologically, heredity here appears to play no rôle. Of my 13 cases 7 were males. The women who were attacked had almost all borne children (one 13 times). A predominant implication of the male sex has been frequently emphasized, and this preponderance perhaps had too much weight attached to it in comparison with the previously mentioned implication in cancer of the gall-bladder, in which disease females unquestionably show by far the greater number of cases. The importance of the question consists in the intimate relation of carcinoma of the gall-bladder to cholelithiasis. In opposition to the views of Naunyn, Leichtenstern, G. Hoppe-Seyler and others, this etiologic factor, however, quite apart from the consideration of the implication of either sex, does not appear to occur to quite the same extent in cancer of the ductus choledochus and hepaticus as the material at hand seems to warrant us in concluding. In the autopsy reports of my own cases, only once do we find mention of a stone in the biliary passages (gall-bladder) and once in the intestines (appendix). Positive proof of cholelithiasis could be determined at most in only three of the histories of my patients; in some of the cases in prior and more recent literature the stone was found directly in the region of the neoplasm, that is, in the carcinomatous structure. Twice in my cases abdominal trauma was assumed to be the cause of the disease. The frequent seat of carcinoma of the choledochus in the lowest portion of the duct appears to me at least to favor the view that deleterious action of the intestinal contents may be a cause. In regard to age, only one of my patients was under 50; seven were past 60, the oldest was 72. In no case was the anatomical extension of the disease limited to the biliary organs. Secondary metastatic carcinoma was found five times in the liver, twice in the lung, twice in the duodenum, once in the pancreas, once in the kidney, once in the uterus, and frequently in the peritoneal glands. In regard to the implication of the proliferal lymph-glands, later reports must be used for comparison. In opposition to the general view that this rarely occurs on account of the absolute stasis of bile and the early death (Leichtenstern counts it among the greatest exceptions!), in my experience the development of metastases was quite invariable and profuse. The liver was always enlarged and its tissues coarser; well developed biliary cirrhosis developed in 3 cases. The portions of the ductus choledochus and ductus hepaticus above the obstruction showed, without exception, more or less extreme dilatation. In 7 cases the gall-bladder was markedly distended to the size of a fist. We find in literature some mention of biliary passages thus dilated from which, by puncture, almost a liter of fluid was obtained. In cases of decided enlargement of the gall-bladder, the tongue-like elongation of the liver, corresponding to the gall-bladder (Riedel's lobe), was noted. In 4 of my cases dilatation of the gall-bladder was not present, yet there was no apparent reason for this absence; twice in the autopsy reports the gall-bladder is mentioned as small and showing a simultaneous long-existing cholelithiasis and cholecystitis. Cholangitis (cholecystitis) with a purulent exudate was present in 4 cases; traces or remains of inflammation of the biliary passages were much more common. Diffuse purulent cholangitis of the finer branches of the hepatic duct (Ely) I have not seen. A few isolated cases of multiple abscess of the liver are mentioned in literature (van Gieson). The adhesions (and ulcerations) observed by me are of such a character as to require no detailed description. None of my patients succumbed directly to perforation, formation of fistula, or rupture of the biliary passages. Dilatation of the pancreatic duct and connective tissue proliferation in the head of the pancreas I saw repeatedly. In the majority of cases (even without other complications) the spleen was distinctly enlarged. Ascites as well as anasarca is mentioned expressly six times in the autopsy findings; in reality it is probably more frequent. Hemorrhage from the neoplasm itself I have observed only once; the hemorrhagic diathesis is mentioned in 4 cases. Appendicitis was diagnosticated in one case and the abscess was opened. At the autopsy the necrosis of the appendix was discovered to be due to a gall-stone (cholesterin). In 3 cases sepsis of the oral cavity (tonsillar abscess, parotitis) was a sequel.

Primary cancer of the duodenum is, as already stated, extremely rare (we are here only interested in that form with a *periampullar* seat; the *parapyloric* form (Nothnagel), that is, cancer situated above the diverticulum of Vater, I shall not describe at all). The former may develop upon the base of a peptic ulcer. This is either the round form of ulcer about th size of a silver quarter of a dollar with medullary infiltrated borders nea the mouth of the ductus choledochus, or the same base, in the center o which a polypus-like protruding portion of mucous membrane is still pre served which carries the ostium of the duct. Such carcinomatous growths, naturally, do not form palpable tumors. The constriction of the ductus choledochus and Wirsung's duct, under these circumstances, is extreme. Extending further, the tumor mass may directly occlude the ducts. The choledochus is dilated to the size of the small intestine, the hepatic duct to the diameter of the thumb, and the gall-bladder attains the size of two fists. The duct of Wirsung is similarly dilated, so that it stands forth prominently, and the smaller lobes of the ducts resemble cysts. In contrast to this there are also cauliflower-like and submucous duodenal cancers, which, on account of their size, may be palpated during the life of the patient. These latter tumors are prone to necrosis and hemorrhage. As long as the growth does not rupture, there is occlusion of the ostia; later, the bile and pancreatic juice may find an outlet. The mouth of Wirsung's duct is dilated and surrounded by shreddy, secondary growths; the same is true of the mouth of the common bile duct. The biliary passages and the pancreatic duct appear to be only moderately dilated. The mesocolon is drawn toward the growths, the intestinal wall is perforated. The head of the pancreas is, as a rule, inseparably attached to the base of the growth.

A case of *cicatricial duodenal stenosis* which I have observed is as follows:

A day laborer, aged 40, who, since his 20th year, had suffered from digestive difficulty and vomiting. In October, 1896, he was suddenly seized with chills and spasmodic pains in the right hypochondrium, and immediately after there was a yellow discoloration of the skin as well as a brown color of the urine. After a brief period the jaundice cleared up. Two months later he had another attack, and at the end of December a third and severe attack lasting 10 days. Since the 2d of February, 1897, his condition has been stationary. He now has pain in the right hypochondrium; the feces show no bile. Occasionally there is vomiting. Fever (up to 102.2° F.) until the 12th of February; later it disappeared entirely. Marked emaciation. Extreme jaundice. No edema; abdomen occasionally shows meteorism. No collateral circulation. Ascites absent during the entire course. Liver is indurated, border readily palpated, surface smooth. Gall-bladder not palpable. Feces show no bile and are rich in fat. Urine contains profuse amounts of bile. Attacks of pain constantly recur.

March 10th, epistaxis. March 14th, right-sided pneumonia. March 17th, vomiting of brownish-black masses; sub-normal temperature. March 23rd, death.

Autopsy: Stenosis cicatricosa duodeni cum occlusione ostiorum d. choledochi et Wirsungiani accidente cholestasi et ictero. Perforatio vesicae felleae supra hepar. Abscess. chron. choliferus. Dilatatio et hypertrophia ventriculi. Peritonitis diffusa.

The stricture had established itself at the site of an old *fistula from gall-stones*. Stones were still present in the common bile duct. This case illustrates drastically the natural cure of incarceration from stone.

DIAGNOSIS; DIFFERENTIAL DIAGNOSIS

After having studied the special pathology of circumscribed adhesive perihepatitis in the porta hepatis, and the affections of the periportal lymph-glands, carcinomata of the head of the pancreas, primary and secondary tumors of the biliary passages, as well as cancer of the duodenum, we shall now turn to the *differential diagnosis*; first, of occlusion of the ductus choledochus due to *stone* and to *tumor* (of the biliary passages themselves and of the neighboring organs). In enumerating and describing the individual diagnostic factors, I shall utilize the most recent literature of the subject (the earlier literature is of little value), and, on account of numerous contradictions, I shall base these remarks upon my own experience. A special classification of the differential symptoms is hardly possible, nor is it necessary; the simplest division appears to me to be indicated by the method of clinical examination. On account of the rarity of obstruction of the choledochus, not only the usual diagnostic factors must be considered, but the exceptions also must be carefully noted; we must guard against schematic force!

Let us begin with the *history*. In the diagnosis of cancer of the choledochus, Leichtenstern lays special stress upon the fact that the affection is prone to occur in *women*. This coincides with the reports of numerous authors, but even Courvoisier has disproven it. My own experiences (see above) also conflict with this statement. Cancer of the pancreas probably attacks men more frequently than women. Bohnstedt maintains that while those persons affected by incarcerated stones usually attribute their malady to a definite trauma which they have sustained, patients wit cancer report nothing of the kind. This does not coincide with my ex perience. The majority of my patients with obstruction due to tumor hav attributed the sudden appearance of the affection to some error in diet, trauma, or something similar. Courvoisier has proposed a diagnostic law *Preceding colic*, particularly if combined with jaundice, is sufficient ev dence for assuming an occlusion of the ductus choledochus due to ston The repeated and frequent appearance for years of one, usually *bri* *attack of jaundice*, especially if it be accompanied by colic, or have pr ceded colic by a short time, is one of the most infallible signs of chol lithiasis. The previous passage of small stones occurs only in occlusi due to calculi. Colic was a symptom in 80 cases of stone in the comm duct, and in 51 of the cases collected by Courvoisier; in 26 cases of p ceding, repeated, brief attacks of jaundice, colic was noted 22 times; 74 cases of obstruction of the choledochus due to other causes, colic reported only 4 times (in two of these last mentioned cases the previ passage of calculi must be considered). Naunyn also declares it to the rule that incarceration of stone is preceded by many attacks of c

According to my observation, typical chronic occlusion of the choledochus due to stone certainly develops after prolonged gastric difficulty (epigastric pressure, spasm of the stomach) sometimes lasting for years; attacks of pain without jaundice first occur, then well-developed colic with fever and violent vomiting, but still without jaundice. This is then followed by jaundice, the intensity of which varies decidedly, and which, after a longer or shorter period, may show complete intermissions. Chronic gall-stone jaundice, therefore, originates in an attack of colic. Colic recurs, but, with every attack, it becomes less severe, the pains decrease, and only chills and jaundice, or the latter alone, remain. Many patients declare that with the appearance of well-developed chills and of permanent jaundice the character of the pain changes. Exceptionally, however, the pain and the other signs of an attack of cholelithiasis in occlusion due to stone may be entirely absent (in Courvoisier's cases their absence is expressly mentioned 10 times), and jaundice may appear without any indication of colic. Various subjective symptoms may then have preceded, but this is not necessarily so. I saw a case (confirmed by autopsy, which will be again mentioned) in which the patient (Ru.), without prodromes, some time prior to his admission to the hospital, and in consequence of an error in diet, was attacked with vomiting, diarrhea and chills; this was followed in four days by moderate sensitiveness to pressure in the right hypochondrium and permanent jaundice. If we ask whether *pain*, and of what nature, usually accompanies the development of tumors leading to occlusion of the common gall-duct, Leichtenstern maintains that, as a rule, especially in cancer of the biliary passages, cholelithiasis (colic), which may, perhaps, have preceded for years, is a factor. This does not agree with the experience of other authors nor with my own. Undoubtedly there are cases of obstruction due to tumor in whose history, even during the entire course of the affection, pain was scarcely ever, if at all, present; that is, there was at most only tenderness on pressure in the gastrohepatic region. In my cases the absence of pain or attacks of pain only prior to the appearance of jaundice is mentioned in 8 cases; in the remaining course of the disease in 5 cases. Among these, unquestionably, there must have been cases of primary neoplasm of the ductus choledochus. Among my 24 cases cholecystolithiasis is proven by autopsy five times, and by typical attacks of colic (exclusive of actual tumors in the biliary passages) three times. Therefore, pain of a varying character is generally, as I believe, much more frequent than its entire absence, particularly at the height of the disease. If Kehr maintains that, in occlusion of tumors, colic is rare, much depends upon what is included under the term "colic." The intensity of these subjective difficulties, before and during the permanent jaundice, naturally varies greatly, and shows all the degrees between an indefinite, disagreeable sensation of pressure in the gastrohepatic region, and boring, lancinating and spasmodic pains of intolerable severity which rob the patient of his sleep, make every position he may assume unbearable, compel him to cry aloud and to long for operative relief, etc. There are cases in which continuous pain predominates; in many others exacerbations are conspicuous, the pain comes in paroxysms, and the attacks are of varying duration. Not infrequently nausea is combined with the pain. An error in diet may produce the first, which is then followed by jaundice, and gastric difficulties not rarely introduce the series of symptoms; subsequently the paroxysms may follow meals. *Vomiting* (vomiting of bile) is mostly absent, that is, such as would correspond to typical gall-stone colic at the height of the attack. Severe, frequent vomiting, often bilious in character, which persists for long periods during the course, is invariably found in duodenal and also in pancreatic cancer when stenosis of the duodenum takes place. The *deep-seated, neuralgic pain, chiefly localized in the region between the pit of the stomach and the navel, radiating toward the back,* and usually referred to the *solar plexus,* is an important diagnostic factor only in certain cases (compare the history of the two cases quoted above). The pains arising from the biliary passages themselves also usually radiate to the gastric region, to the back, and especially to the shoulder. I am inclined to attach especial value to this *shoulder pain* in the diagnosis of affections in the region of the biliary passages, but I have never succeeded in making a positive differentiation between occlusion of the choledochus by a neoplasm of this duct or by a pancreatic cancer from this sign alone. I attach a certain significance to the circumstance that, in cancer of the duodenum, the pains increase afte the ingestion of food. But this is a differentio-diagnostic factor of bu relative importance, in that pains in obstruction due to tumors of th ductus choledochus only exceptionally correspond to typical colic (*pain and vomiting*) of gall-stone (in which, of course, I leave entirely out o consideration secondary carcinoma of the ductus choledochus in primar cancer of the gall-bladder).

In the diagnosis of cholelithiasis, Neusser and Ortner attach great ir portance to the *jaundice* which *in a brief time becomes marked,* that i soon reaches its *maximum.* A villous cancer growing rapidly may al in a short time give rise to extreme icterus. In carcinoma of the du denum, the appearance of jaundice is said to be much more gradu In a series of cases of carcinomata of the pancreas Leichtenstern fou that icterus appeared in an acute form and in a few days reached maximum. I attach but little weight to the statements of the patie which are often quite vague, as to the manner in which the perman jaundice appeared. I know of undoubted cases of incarceration of sto in which jaundice developed gradually; this seems most apt to occur cases in which the stone is partly in the cystic duct, and partly in common duct. Among my own cases of obstruction due to tum 5 times the history showed that jaundice had appeared "suddenl in a few hours or in a night, and this was also true in cancer of the pancreas.

Another, perhaps significant, diagnostic difference between the development of permanent jaundice from obstruction due to calculi contrasted with occlusion from tumor is the *timely extension* of the same. This factor is naturally considered with the history, and in the further observation of the case. Here again Courvoisier was foremost with the definite statement that permanent jaundice lasting several years generally indicated stone and excluded any other form of obstruction. He mentions cases of obstruction due to stone lasting 2 to 5 years, and even longer. Kehr operated upon and cured a patient who, for 12 years, suffered from the symptoms of occlusion by stone. Schüppel, as well as Neusser and Ortner, on the other hand, exclude cancer in general (except, possibly, primary carcinoma of the gall-bladder) if the jaundice persists longer than 6 months. This latter period, however, appears to me to be entirely too limited. In my cases of primary cancer of the biliary passages, the total duration of the course from the onset of chronic jaundice up to the time of death was 7 to 9 months; in one case only was it less than 6 months; in my cases of cancer of the pancreas, 3 lasted less than 6 months, and in all the others the duration was 8 to 11 months; in duodenal cancer the course was somewhat longer than 6 months. Perhaps the average duration in cancer of the head of the pancreas is slightly less than in cancer of the choledochus. With Naunyn and others, I think it wiser to estimate the duration of jaundice which excludes cancer at about one year. In practice, however, there is no absolute, precise law. For, in occlusion due to stone, death may occur also after a brief duration of the jaundice, often a few months, often only weeks or days! In most of the known cases, death was preceded by symptoms of hepatic autöintoxication from the 6th to the 12th month; therefore, chronic gall-stone jaundice rarely lasts longer than one year. The reason why occlusion of the common duct by a tumor produces jaundice which lasts such a short time is, according to Courvoisier, simply this,—that here the underlying disease in some other manner early causes death. But I do not believe this. I am inclined to agree with Kehr, and admit a chronic closure of the common duct by stone in the supraduodenal portion, and one due to calculus in the vicinity of the duodenal papilla. In the latter case, and under severe circumstances, the jaundice is at once intense, and varies but little from that observed in most obstructions due to carcinoma. In these later cases, however, provided a choledocho-duodenal fistula does not bring relief, or if a choledochotomy is not performed, a fatal termination is just as certain and as rapid as in carcinoma of the common duct and pancreas, and with the same symptoms of severe inanition and hepatargy. In no less than 14 of my own cases, without regard to the focus for the development of the cancer, this was absolutely demonstrated (often by prolonged coma with or without the hemorrhagic diathesis). Among auxiliary causes of death suppuration was found in four cases, an old syphilis in one, portal vein stasis in another, and in a few, metastases.

Jaundice, as a cardinal symptom, occupies a leading position in the status and course of the clinical history. It may be assumed from the onset that a stone with its changeable form, or even a great number of stones, will not completely occlude the lumen of the bile ducts, as would, for instance, be the case with a soft tumor which perfectly adjusts itself to the lumen and the wall of the canal, and entirely occludes the former. Even Cruveilhier attempts to base this theory of constriction, *a priori,* upon the comparison of the pathologico-anatomical changes which occur in incarceration from stone. Behind the stone arrested in the common bile duct stasis of bile occurs, whereupon the passage dilates, the lumen increases to the circumference of a finger, or even that of the small intestine. Then the bile rises to the stone, floats in the dilated common duct, and passes it. The jaundice may then entirely disappear. But the stone invariably floats along to lower parts of the gall-duct toward the intestine. Clinical experience confirms the opinion that in chronic occlusion of the common bile duct by stone, a *decided variation in the intensity of the jaundice,* even its *complete disappearance,* is very characteristic. Under these circumstances the urine likewise shows variations in color between dark and light, and only in rare cases are the feces permanently free from bile. In cases of obstruction due to tumor, the jaundice is usually intense and does not vary so manifestly. The feces are persistently acholic. In contrast to the usual conditions, however, we must bear in mind that als in incarceration from stone (in the papilla of the duodenum) the complet exclusion of bile from the intestine, with all its serious consequenc (hepatic intoxication, the hemorrhagic diathesis, and death in from 6 t 12 months) is quite possible, and this may occasionally occur even whe permanent jaundice has been the first alarming symptom of a cholelithias previously latent. An example of this kind is my case before referred (Ru.). Here for a short time only the jaundice was moderate, the fec slightly tinged with bile, but the symptoms rapidly became more inten and the feces subsequently and during the 7 months of the protract course of the disease were absolutely acholic; the course was that sev form just indicated. Moreover, in my experience it is the rule that a in cases of obstruction due to tumor, variations in the intensity of jaundice are quite manifest, but they are never very decided ones. P ticularly at the onset of the affection, paroxysmal aggravation of all subjective symptoms, produced by a still more marked impediment the flow of bile, brings about a renewed staining of the visible m branes. Haarmann believed that a complete but temporary disappeara of jaundice in obstruction from tumor could be explained by the dest tion, and its recurrence by the renewed proliferation, of a soft tu which in his case was situated at the ostium. Courvoisier admits the probability of similar conditions from analogous observations by Bourceret and Bradbury. Neusser and Ortner designate as a decisive factor in the diagnosis of carcinoma of the common bile duct the circumstance that, in the course of a jaundice which has developed to its maximum intensity combined with acholia of the feces (usually after a duration of from 2 to 3 weeks) a full, even abnormally profuse, biliary staining of the feces may again occur; this may happen once or repeatedly, suddenly or at least rapidly, occasionally with fulminant symptoms; there may be a slight admixture of blood to the dejecta or to the vomitus; in a brief time, the condition gives place to a new period of acholia. I have reason to believe that instances of this kind frequently occur. In one of my own cases of primary cancer of the choledochus (at the onset of permanent jaundice) the previously intensely yellow discoloration of the skin decidedly diminished, and the white, clay-like stools again became tinged with bile; this change lasted for only 8 days, and was followed by an aggravation and permanent acholia which was unyielding. Hemorrhages from cancer of the common bile duct, even to a marked extent (black feces), are not uncommon. One of my cases of cancer of the duodenum also exhibited the condition described by Neusser, and an anatomical preparation satisfactorily explained it as due to decided ulceration. Naunyn also mentions a case of Coupland's of neoplasm in the duodenal wall at the diverticulum of Vater, in which a disappearance of the jaundice, biliary staining of the feces, and a simultaneous decrease in the size of the distended gall-bladder could be repeatedly recognized. In two of my cases of occlusion of the choledochus from tumor of the pancreas, besides a manifest and very varying jaundice of the skin, there was long-continued diarrhea, and feces containing bile, some showing deficiency of bile, and completely acholic stools alternated. Finally, cases of obstruction from tumor exist (two among my own) in which the feces up to the time of death show only a feebly feculent character. Somewhat different from the variations in the intensity of the jaundice in incarceration from stone and occlusion from tumor, which have been described, is a clearing of the skin and urine after a long continuance of the jaundice without the feces losing their clay color. The significance of this prognostically unfavorable sign is evident from the foregoing descriptions.

In the opinion of different investigators the diagnostic *importance of fever* varies. By many the presence of fever preceding and during the permanent jaundice, and especially the repeated occurrence of chills at regular or atypical periods, combined with attacks of pain, with sudden rises of temperature, and, later, also the remittent or continuous rises of temperature in chronic obstructive jaundice are referred either to gall-stones, that is, to suppuration, to ulceration, etc., in the biliary passages (the liver) or to a superadded pyemia. Courvoisier reports that in 80 cases of occlusion by stone 19 (therefore about $\frac{1}{4}$), and of 74 obstructions due to other causes 7 (about $\frac{1}{10}$), were accompanied by fever. I must emphasize here that I also know of cases of impacted stone which, in certain phases of the affection, showed jaundice only some time after the attacks of fever (previously frequent) had ceased entirely. Leichtenstern maintains that as soon as cancer of the gall-bladder or of the biliary passages appears the preceding inflammatory phenomena due to gall-stones cease entirely. High fever or chills are said categorically to contraindicate the presence of cancer although numerous other symptoms may be present which favor it. According to Kehr, in occlusion by tumor fever occurs in exceptional cases, particularly in the last stages of carcinoma. In the case of scirrhus of the pancreas this author has observed multiple abscesses in the liver and perihepatic suppuration. Naunyn emphasizes the fact that fever combined with chills is observed with especial frequency in neoplasms of the liver (even those due to metastases). In 15 of my cases of occlusion due to carcinoma, there was fever lasting a long or short time. Only 5 times, either clinically or at the autopsy, was it possible to attribute it to a complication outside of the biliary passages themselves (parotitis, tonsillar abscess and pyemia, cystopyelitis, suppuration of the glands of the neck, gall-stone appendicitis). Fever, with either a moderate, intermittent or remittent course or (not so frequently) with decided rises in temperature to 101.3° F. and 103.1° F., was present. In the late stages of the disease (leaving out of consideration the closing days of life, when lobular pneumonias, etc., developed) the fever usually but not invariably became more marked. Periodically recurring or even isolated chills I hav never observed (except in a case of lithogenous cancer of the gall-bladde which proliferated into the biliary passages). I am unable to decide whethe fever is more frequent in that form in which there is proliferation int the duodenum leading to destruction, which is particularly the case i carcinoma of the head of the pancreas, or in primary cancer of the biliar passages. As will be observed, I can take only a middle position in regar to the differentio-diagnostic value of fever; all the more so since, i literature, I note that chills also occur in occlusion due to carcinoma.

The *degree of inanition* and the *loss of weight* in all of my cases tumor obstruction have been very considerable; *cachexia* developed wi exceeding rapidity. I have not observed that cachexia præcox is peculi to cancer of the pancreas; nor have I noted subnormal temperature in su cases (the syndrome of Bard and Pic). In my cases the N-excretion comparison to the intake was not determined. In doubtful cases, amo other signs, light was occasionally shed on the diagnosis by the estimati of N and NaCl in the urine (diminished excretion of chlorin in cance I have no personal experience of the proportions of N and P_2O_5 in excretion of persons suffering from obstruction of the choledochus tumor, nor in regard to the increase of ammonia and the separation the total nitrogen into the various N-containing products of the urine. In all of my cases of obstruction by tumor in which an examination was made, deviations from the normal blood were noted, although not very decided: decrease of hemoglobin to 75–65 per cent. (Fleischl), decrease of the red blood-corpuscles to 5,800,000, 4,100,000, or 3,900,000; the number of white corpuscles (not directly due to the fever) varied between 5,800 and 16,400. In one case (carcinoma of the pancreas with enteritis and fever) very decided oxyphilia of the blood was observed. Finally, we should be most cautious in drawing far-reaching differentio-diagnostic conclusions from the miserable appearance of the patient and his loss of weight!

In discussing biliary stasis due to obstruction of the choledochus I have not minutely considered the dilatation of the biliary canal. In the first place, experience shows that dilatation occurs in the common and hepatic ducts. Particularly in cases of closure near the opening of the ducts into the intestine do dilatation and elongation occur, and these ducts may enlarge to the size of a thumb or a coil of the small intestine. The common duct occasionally forms a sort of cyst, protruding from under the liver, containing from one to several liters of bile, and has, in fact, been confounded with the gall-bladder upon which operation has been attempted. The dilatation of the final branches of the hepatic duct extends usually to the periphery of the liver, and may produce fluctuating protuberances on the surface of this organ, or, upon a uniform dilatation of the intrahepatic canals, the liver *in toto* becomes enlarged, but, as a rule, it shortly after again decreases in size. In chronic obstruction of the common duct *dilatation of the gall-bladder* and of the cystic duct does not occur with the same frequency. As we were inclined to believe from the reports mentioned in text-books, Courvoisier found the tissue changes in the gall-bladder different and more complicated. Previously it was the general opinion that permanent closure of the choledochus invariably led also to a dilatation of the gall-bladder. Courvoisier found, however, that among 109 fully described cases of dilatation of the gall-bladder, 17 only were due to occlusion by stone; all other obstructions of the ductus choledochus were due to different causes (10 to tumors in the interior, 8 to obliterations, 74 to external compressions). Furthermore, the dilatation of the gall-bladder in obstruction from stones was usually of only moderate extent, and in but a few cases was the organ stated to be enormously enlarged. On the other hand, dilatation due to other causes of occlusion was often very decided, and in some cases colossal tumors were formed. In contrast to these dilatations are 78 cases also collected by Courvoisier, in which, in spite of occlusion of the common duct and a patulous cystic duct, the gall-bladder was not enlarged but atrophic. Of these, 70 (i. e., 90 per cent.) were cases of impaction of stone in the common duct, 8 only (i. e., 10 per cent.) were due to other causes (in 5 of the last-mentioned cases the gall-bladder also contained stones). Courvoisier separates his 187 cases into two groups, one containing 87 cases due to obstruction from stone and 100 from other causes of occlusion. With incarceration of calculi, atrophy of the gall-bladder occurred in 80.4 per cent., dilatation, which was found in 19.6 per cent. of the cases, being much rarer. The other causes of obstruction, on the contrary, much less often ran their course with atrophy (8 per cent.), dilatation being much more frequently present (92 per cent.). In impaction of stone in the common duct, therefore, dilatation of the gall-bladder is rare, the organ being previously contracted by connective tissue (chronic cholecystitis, atrophic effect of adhesion). In occlusion of the choledochus of other nature, however, dilatation is the usual result. Courvoisier collected 35 cases of obstruction of the common duct in which operation had been performed; in 17 cases due to stone 4 only showed dilatation, and in 18 due to pressure from tumor, from stricture, etc., dilatation was found in 16; of the first 17 there were 13, and of the last-mentioned 18 only 2, without enlargement of the gall-bladder. Of the 20 cases of dilatation of the gall-bladder, 4 only were due to obstruction by stone, 16 being from other causes The correctness of this view has been confirmed in a large number o cases by subsequent investigation. Encklin (1896), in a total of 17 cases of occlusion due to stone, found contracted gall-bladder presen in 64 per cent.; in 19.7 per cent. the organ had retained its normal siz and in only 16.25 per cent. was it dilated. On the contrary, in 129 cas of occlusion from other causes (stricture of the choledochus, compressio of the duct by enlarged lymph-glands, tumors of the common duct and the pancreas) the gall-bladder was found dilated in 87 per cent., a contracted or normal in only 6.5 per cent. Courvoisier regarded t incontestable contraction of the gall-bladder from the presence of sto in the common duct a particularly valuable point of support in the diff ential diagnosis. Naunyn, Kehr, Langenbuch, Bohnstedt, and others c curred with him. This diagnostic point is referred to (particularly French literature) as *Courvoisier-Terrier's symptom.* Terrier, howev only followed Courvoisier's teaching. G. Hoppe-Seyler maintains a c servative position. Leichtenstern, "naturally," does not conform to Courvoisier rule in deciding the question whether occlusion of the com duct due to stone or cancer of the biliary passages is present, the la of which he declares to be lithogenous; he maintains absolutely tha primary carcinoma of the choledochus there is no enlargement of gall-bladder, while, in contrast to this, in cancer of the pancreas a filled gall-bladder can be determined. Neusser and Ortner declare in diagnosis of primary cancer of the ductus choledochus that a disti enlarged gall-bladder is of "little importance." Quénu also maintains the Courvoisier system is not absolutely decisive, and reports some mis in diagnosis.

I have already briefly alluded to the results obtained in my autopsies. In my opinion, in a diagnostic respect (I am considering only the diagnosis prior to operation), and without weighing the autopsy findings, the question is as follows: Are the cases with a non-palpable gall-bladder in obstruction due to tumor, and the cases with an enlarged palpable gall-bladder in occlusion due to stone, actually so rare that Courvoisier's sign may be looked upon as pathognomonic, and that, in each individual case, a decision may be made from this? In my 8 cases of primary carcinoma of the choledochus, during the entire course of the disease the gall-bladder was 4 times non-palpable, and in my 7 cases of cancer of the head of the pancreas with occlusion of the common duct, this was the case 3 times. In one instance of primary cancer of the gall-bladder with subsequent cancer of the choledochus, the gall-bladder could not be felt. In one case of duodenal carcinoma the gall-bladder was visible, in the second case it could never be seen. The gall-bladder was observed to be anatomically dilated in some cases in which the enlargement could not be discerned clinically. The largest tumor of the gall-bladder which was found during life in my cases was about the size of an apple. The tumor invariably had the characteristics of the tumors of the gall-bladder which have been described. Fluctuation at the top of the tumor was never absent. In two cases with particularly well-developed Riedel's lobes it was readily perceived that a neoplasm had formed at the point of transition into the coarser liver. In a clinical history pain upon pressure of the tumor is sometimes emphasized, sometimes absent. The growth of the tumor and its variation in size have been occasionally mentioned. In 4 positive cases of obstruction due to stone I remember that the gall-bladder was more or less enlarged to palpation. In one of these cases, we were able during the life of the patient to determine that the gall-bladder was not absolutely smooth and freely movable, as is the rule in occlusion due to carcinoma. In my opinion, in cholelithiasis much depends upon how and when the impaction of stone takes place. In a case repeatedly mentioned (Ru.), in which permanent jaundice was the first alarming symptom of cholelithiasis, and the cystic duct was not obliterated, the gall-bladder was very markedly dilated, yet no stone was present in the cystic duct, and, therefore, in addition to the obstruction of the choledochus, there was neither dropsy, empyema of the gall-bladder, nor obliteration of the duct. Courvoisier collected 12 cases from literature in which the last-named accident supervened. Kehr also calls attention to the diagnostic difficulties when we have a combination of dropsy or purulent cholecystitis of the gall-bladder with chronic occlusion of the choledochus. He operated on 5 cases of this kind. In the differential diagnosis between occlusion of the choledochus due to stone and to tumor, after all that has been said, and apart from the fact that the exceptions also must be considered (on account of the rarity of the cases) no actual pathognomonic importance can be attributed to the Courvoisier symptom, the value of which, I believe, is often exaggerated.

According to Naunyn, there is often no marked *enlargement of the liver* in chronic gall-stone jaundice. With obstructing carcinoma of the common duct and of the pancreas, also, the liver is usually small (for cancer of the gall-bladder, Bard and Pic hold similar views), while in cancer of the gall-bladder, as a rule, decided enlargement and nodular consistence of the liver are never long absent. In my clinical histories of tumor occlusion, enlargement of the liver is frequently and particularly emphasized. In 5 cases, metastatic cancerous nodules could be palpated on the surface of the liver during the life of the patient; anatomically, metastases of this organ have been even more frequently proven; upon examination, the liver may appear smooth although carcinomatous nodules have already formed. Considering our previous remarks on this subject a minute examination of the liver for secondary cancerous nodules (and also palpation of the cavum Douglasii) should never be omitted. When carcinoma of the liver is absent, the consistence of the organ is, as a rule, moderately increased. Biliary cirrhosis was twice anatomically determined with certainty during the life of the patient.

Most authors agree that, in the majority of cases, the *tumor* which causes obstruction, as such, notwithstanding exact and repeated investigations, is not palpable. I was able to palpate it in only one of my cases of carcinoma of the pancreas and in one of my two cases of cancer of the duodenum (the latter only occasionally). Leichtenstern describes a tumor at the head of the pancreas which may generally be definitely palpated; carcinoma of the duodenum below the papilla he diagnosticates by the presence of a tumor, etc. Moncorgé emphasizes the presence of a tumor in the median line. In the case of tumors of the pancreas leading to obstructive jaundice, I was unable to confirm this. In malignant tumors which develop in an area insusceptible to palpation, G. Hoppe-Seyler expects results from the X-rays; but I cannot share this hope because of my experience in other cases.

The consensus of opinion (Naunyn, Kehr) is, that *ascites* is absent in uncomplicated jaundice due to stone. In my case (Ru.) it was present as well as anasarca and biliary cirrhosis, without peritonitis and without thrombosis of the portal vein. On the other hand, in neoplasms of the biliary canals and in carcinoma of the pancreas, ascites is not rare. Excepting one case of cancer of the pancreas in which aspiration was necessary, I found slight ascites 10 times, and anasarca 6 times.

Naunyn reports that *splenic tumor* is rare in carcinoma; on the other hand it is common in chronic gall-stone jaundice (not merely in addition to diffuse hepatitis!). In 8 of my cases of obstruction due to tumor there was decided prominence of the spleen, and in 4 it could be palpated. I do not believe the condition of the spleen of paramount significance in a differential diagnosis. According to my experience, in any chronic jaundice (even in catarrhal jaundice apart from cirrhosis), the spleen may be enlarged; and, moreover, the enlargement of the spleen may also be explained by a superadded stasis of the portal vein (pressure of the tumor upon the trunk or branches).

According to Leichtenstern, a sign which is of great importance in the diagnosis of cancer, although not very frequent, is that the malignant infiltration causes a *swelling of the "jugular gland"* (Virchow's gland), a lymph-gland situated behind the clavicular insertion of the sterno-cleidomastoid. Laterally this swelling may often extend above the muscle, may attain the size of a cherry, a chestnut, or, occasionally, of a hen's egg; it is invariably present on one side, generally the left, is occasionally isolated, i. e., with no other enlargement of glands in the supraclavicular region or in the neck. Kehr confirms Leichtenstern's observations; but the gland is found only in advanced stages of the disease when the diagnosis is no longer difficult. My clinical histories contain exact reports of the enlargement of peripheral lymph-glands; frequently of the inguinal glands, sometimes those of the axilla (particularly upon the right side); in the neck, as a rule, the enlargement is multiple and bilateral, and in the supraclavicular regions; enlargement of the jugular gland alone is mentioned rarely; in one case it was decidedly increased in size and finally broke down.

In the preceding, a series of factors are mentioned which are of varying importance, which, carefully combined and utilized, will facilitate the differential diagnosis between chronic impaction of stone and obstruction due to tumor. Incarceration of stone complicated by carcinoma of the common duct (lithogenous) which occurs occasionally (see above), scarcely permits a positive decision; in such cases, from the standpoint of professional treatment only the diagnosis of cancer is necessary. Stricture of the common duct following cholelithiasis is not always characterized clinically in the picture of the disease. In such cases, on the assumption that stones are present, I advise operation, and in case no stones are found, operative procedure is to be limited to the breaking up of adhesions, etc., which is of marked, although only temporary, benefit. Complete obliteration of the choledochus, provided intense permanent jaundice (with enlarged or nonpalpable gall-bladder), repeated attacks of jaundice of brief duration, or typical colic with fever have not preceded, may be referred to cholelithiasis; and in a differentio-diagnostic respect, ulcer of the duodenum (cicatricial stenosis of the duodenum) and obstruction due to cancer must be principally considered.

Up to the present time, the only diagrammatic methods for the grouping of the different forms of occlusion of the biliary passages not due to stone have been short formulas proposed by Neusser and Leichtenstern; Nothnagel also furnishes such for the duodenum, Oser for cancer of the pancreas.

SYMPTOMS IN FAVOR OF PRIMARY CANCER OF THE COMMON GALL-DUCT

According to Neusser	*According to Leichtenstern*
A duration of the disease beyond six months excludes cancer. Decisive negative signs: Absence of tumor upon palpation. Positive factors: Suddenly during permanent jaundice and while there is an admixture of blood to the feces, complete biliary staining of the feces is seen, rapidly changing again to acholia. Eventually infectious cholangitis. Prolonged absence of gastric symptoms. Less important: Distinct enlargement of gall-bladder.	Aged people, particularly women. Cholelithiasis, even though years previously. No palpable tumor. Metastases extraordinarily rare. No fever. Painless melasicterus of long duration. Acholia of the feces. Liver large and smooth. No tumor of the gall-bladder. Cachexia. Anasarca. Eventually: Enlargement of Virchow's gland.

SYMPTOMS IN FAVOR OF CARCINOMA OF THE PANCREAS AS A CAUSE OF OCCLUSION OF THE COMMON BILE DUCT

According to Neusser	*According to Leichtenstern*
Besides jaundice: Celiac neuralgia; from the onset decided aortic pulsation in the epigastrium, and the absence of a localized, or at most an indistinctly palpable, tumor. Later glycosuria (maltosuria), the absence of indicanuria; profuse amounts of transverse striped muscular fibers; undigested starches, deficiency of fat-splitting in the feces.	Intense jaundice, acholia of the feces. Occasionally a tensely filled gall-bladder. Pancreatic tumor usually distinctly palpable. Persistent and severe gastric symptoms. Late disappearance of free hydrochloric acid from the gastric contents.

SYMPTOMS IN FAVOR OF CARCINOMA OF THE SMALL INTESTINE BELOW THE DIVERTICULUM OF VATER

According to Neusser	*According to Leichtenstern*
Jaundice; acholia of the stools, temporarily disappearing and then recurring; a gradual change in, perhaps even gradual first development of, the jaundice. No marked symptoms of pyloric intestinal stenosis, but other dyspeptic symptoms without recognizable disturbances of the gastric chemism. Melena without hematemesis. Late: Lumbar pains. Slow course. Carcinomatous metastases often absent (liver).	Rarity of this carcinoma. Duodenal cancer early causes stenosis of the intestine, in consequence of which massive bilious vomiting and gastrectasis occur. Tumor. Conspicuously acute development of the jaundice.

In regard to my own views, in several points I must agree, and very frequently disagree, with these contradictory statements, and my position may readily be determined from the foregoing explanations. One point only I shall briefly touch upon. In regard to the presence of gastrectasis, which is maintained by Leube in those cases of duodenal cancer in which the neoplasm is situated in the diverticulum of Vater, I concur in the opinion of Neusser. Typical, graphite-like fatty stools, i. e., such as are about one-half composed of fatty acid needles, are mentioned in only one of my cases. In regard to fat-splitting in the intestine, I, unfortunately, undertook no special investigations. As, however, according to experience, in carcinomata situated at the mouth of the ductus choledochus there is sometimes a simultaneous occlusion of the duct of Wirsung, and in consequence an exclusion of the pancreatic juice from the intestine leading to dilatation of the pancreatic duct and even to connective tissue proliferation in the tissue of the pancreas (I saw this in two cases), I do not expect such examination of the feces to aid us much in the differential diagnosis. In all of my cases there was either constipation or diarrhea. Glycosuria upon ordinary diet I did not once observe in my cases of carcinoma of the pancreas. In my recent cases, including the cases of cancer of the pancreas, the systematic test of the patient's power of assimilation for grape sugar never showed alimentary glycosuria beyond the ordinary amount; in only one case was there marked glycosuria with other signs of diabetes even on an albumin diet. This was a positive case of carcinoma of the ductus choledochus, with a metastatic, malignant involvement of the lymph-glands in the concavity of the celiac plexus, and slight hardening of the pancreas. Indoxyl was sometimes present in the urine, sometimes absent, but not only in the pancreatic tumors. Polyuria was an inconstant finding in my cases of cancer of the pancreas. Finally, but little reliance can be placed, diagnostically, upon the secretory function of the stomach: in two instances of cancer of the biliary passages, and in one of cancer of the pancreas, free hydrochloric acid was absent in the gastric contents. In one of the cases of duodenal cancer the reaction for free HCl was positive. The motor function of the stomach was not conspicuously impaired.

In conclusion, a few words concerning the differential diagnosis in the case of *the new-born.* Here intense jaundice, lasting longer than 14 days, with absence of bile from the feces, points to a serious disease of the large gall-duct. It cannot be merely a case of syphilis of the liver, but is due to the congenital absence, that is, fetal occlusion, of the biliary passages.

Our reason for making an early diagnosis minutely in cases of chronic obstructive jaundice is already indicated, but it is by no means, as Bohnstedt thinks, because, with the early recognition of a cancer of the biliary passages, this may be removed by operation. On account of the anatomical ment (melanoangiomata); this, however, does not permit the assumption that a tumor of this kind, or angiomata in general, will become carcinomatous.

Non-parasitic cysts of the liver must be included with the neoplasms because, at least in part, they originate from new structures, newly formed biliary passages; they may all be regarded as originating from the vasa aberrantia or from the intrahepatic biliary passages (related to the adenomata). We usually differentiate solitary, congenital, isolated cysts from multiple cysts of the liver developing during postuterine life. The congenital cysts, as a rule, do not reach great size; in certain cases, however, they become important on account of their decided volume, that is, in consequence of the ascites they produce, or by an obstruction to the birth of the child in question (Witzel, Bagot, Sänger, and Klopp). Multiple cystomata or cystic liver (cystic degeneration of the liver and kidney) may frequently but not invariably be combined with cirrhosis of the organ (also being in causal connection [?]). Besides those the size of a pin's head, there are others which contain as much as a liter of fluid; upon section, a liver of this kind shows a collection of cavities, the smallest of the size of a pin's head, and upon the surface of the organ larger and smaller protuberances form. The liver varies in size; usually it is enlarged. Biliary, portal vein, and even vena cava stasis may be the consequence. In combination with those of cystic liver, analogous changes occur also in the kidneys, to which the symptoms of chronic nephritis may be added. More rarely there are simultaneous cysts of the ovary, the uterus, the seminal vesicles, and of the thyreoid gland. Occasionally, multiple cysts of the liver cause no symptoms during the life of the patient. Sometimes such a liver may be palpated (nodular surface of the organ, fluctuation of the cysts). Puncture of the latter for diagnostic purposes should never be performed, as this induces suppuration; besides, it furnishes no positive facts. The rarer, simpler (that is, isolated) serous or biliary cysts of the liver usually form in advanced age (after the 40th year of life), sometimes earlier than this, and, as it appears, more frequently in women. Their seat is usually the upper, occasionally, however, the lower surface of the liver, immediately beneath the capsule. Their shape is globular or oval, but there is a variation in size which may even attain that of a child's head. The liver may be distinctly nodular, and sometimes the greater part of the cystoma extends above the hepatic mass; in rare cases the cys may develop at the expense of the parenchyma to such an extent tha the capsule and scant remains of tissue form only a thin sac. Suppura tion has been observed. Operation upon such hepatic cysts (the cases i question are congenital cystomata and cystic degeneration) have been performed by Hueter, North, Terillon, Winckler, Kaltenbach, Berg, Baye and others.

In passing to the *malignant* neoplasms, we must first devote a fe words to *sarcomata* which up to the present time have unfortunately possessed greater pathologico-anatomical than practical interest. The occurrence of primary sarcomata was for a time denied; but some quite positive observations (Podroužek) have been recently reported. The focus of the disease is principally the region of the branches of the portal vein, and particularly the wall of the smallest vessels, as these are most apt to show the characteristic proliferation of sarcomatous tissue. At the autopsy of an undoubted case of Kahlden's which may be positively looked upon as primary, in a man, aged 32, who had suffered from ascites, the liver was found permeated by circumscribed, multiple, medullary nodules, the surface of which in many areas was umbilicated, so that they closely resembled malignant metastases. Microscopic examination revealed their size, the older cirrhotic changes, the sarcomatous nature of the tumors, and permitted the recognition of a distinct tendency to alveolar structure. The number of cell forms, as shown by the microscopic examination, also in sarcoma of the liver (primary and secondary), is a very varying one. Clinically, the question of the cell type of sarcoma of the liver is scarcely of importance; the malignity of the change shows only slight gradations. *Melanosarcoma,* although very rarely, also occurs primarily in the liver (Block, Belin and others). But the majority of sarcomata of the liver are, as a rule, secondary growths, and, as is readily conceivable, metastatic sarcomata of the liver are not as frequent as those of the lungs; why this condition is reversed in carcinomata, we shall see later on. Often the primary sarcoma which secondarily infects the liver is situated in an area of the body which may be reached by the physician (in the muscles, bones, breast, etc.); sometimes, however, a careful autopsy shows only a small primary tumor situated, for example, in the apple of the eye. In the case of melanomata the original neoplasm is usually in the choroid or in the skin (sarcomatously degenerated nævi pigmentosi). After the enucleation of the diseased bulb, years may sometimes pass without the liver being attacked. The fact is of practical significance that the skin may also be secondarily diseased. All secondary sarcomata in the liver cause either an extensive formation of nodular tumors or a more diffuse infiltration. The particularly malignant melanomata may be the cause of extremely large tumors of the liver, by far exceeding in size the non-pigmented growth. Occlusion of the capillaries and succeeding thrombosis of the intrahepatic portal branches produce ascites.

Excepting for the longer period of time which, at least occasionally, elapses between the appearance of the primary neoplasm and the discovery of sarcomatous metastases of the liver, the *symptomatology* of secondary sarcoma of the liver hardly differs from that of medullary cancer, that is, of secondary carcinoma of the liver. The sarcomatous metastases of the liver may remain latent during the life of the patient or may become prominent in the symptomatology. The organ enlarges decidedly, its surface is covered with nodules, ascites appears (or may also be absent). Digestive disturbances, pain, and jaundice are frequent.

Melanomata of the liver are sometimes unrecognized in that they are masked by disturbances due to other and more prominent metastases. In other cases the liver affection is the most obvious, that is, it represents the only secondary disease. Then there are digestive disturbances, even vomiting, the strength decreases, the right hypochondrium is markedly sensitive to pressure, dull or sharp pain, increasing in paroxysms, and radiating to wide extent, appears; the volume of the liver increases decidedly. Its surface is by no means always nodulated; it is probably so only in the majority of cases; often it is smooth to the touch, and hard as wood. The right hypochondrium, the epigastrium, and, eventually, the entire abdomen are correspondingly distended. Meteorism and ascites are rarely absent; the spleen, however, is not decidedly increased in size. The stools may be acholic or may contain bile. Jaundice is very rarely present. Moderate fever is comparatively frequent. In all forms of hepatic sarcomatosis this latter symptom may be most prominent; its intensity is then considerable, and, with an intermittent type, chills, etc., may be added. The urine which, upon casual examination, is of normal color, if placed in the light, with an addition of saltpeter or chromic acid, turns black (Eiselt). This melanuria is of great diagnostic significance; unfortunately, however, the symptom is rarely present. The total duration of the disease after the affection of the liver has been determined varies between 1 and 6 months; on the average it is 2 to 3 months.

In by far the majority of cases, even with a tentative diagnosis, for the confirmation of which an exploratory incision may sometimes be necessary, the affection of the liver is so diffuse or manifold that (apart from other metastases of the neoplasm) operative removal cannot be attempted. The question whether a "malignant tumor" in the liver is solitary or multiple can only be answered after the abdominal cavity has been opened. Isolated resections of liver sarcomata have been reported (J. Israel and others).

Tumors of the liver composed of epithelial elements are still the subject of controversy among pathological anatomists, and particularly the relation between *adenoma* and *carcinoma,* although, according to the investigations of Hanot and Gilbert as well as of Siegenbeck van Heukelom, it is very likely that there is a fundamental difference between them; for the present, at least, the rare *solitary tubulous adenoma* of the liver is of special clinical interest because large tumors of this kind have repeatedly been successfully removed by operation. While I shall omit therefore, the *benign, solitary, nodular hyperplasia* of the liver (Rokitansky, Klob, Mohamed and others), I shall briefly discuss the ***multiple nodular hyperplasia*** (which, according to experience, has a certain tendency to adenomatous and carcinomatous formation) as well as the ***multiple*** *tubular adenomata* which I place with the group of hepatic carcinomata (cirrhosis carcinomatosa).

In a case of Keen's an adenoma which was extirpated proved to be a coccidia tumor; perhaps this etiology really plays a rôle (?). In rabbits, at least, there are adenoma cysts arising epidemically in the liver in the papillary proliferations of which Felsenthal and Stumm have found coccidia in various stages of development. As a rule, the solitary hepatic adenoma of man runs a comparatively benign course and does not give rise to metastases. In a few cases, however, the proliferation of the tumor into the portal veins and a thrombus-like growth has been described; in a case of Perls, an actual metastasis with glandular tubules was found in the sella turcica, and from the lumen of the tubules a greenish mass was excreted (Schmidt describes a primary hepatic cancer with metastases, and a nodule in the sternum of similar structure to that of the liver, in which bile was positively secreted); in another case secondary nodules were found in the lungs and in the mediastinal glands. As the hepatic adenomata increase in size, regressive metamorphosis and hemorrhage occur (with the formation of "blood cysts"), occasionally even to suppuration. In literature *cystoadenomata* are reported with a single layer of cylindrical epithelium and serum-like or colloid contents; they are connected with the biliary passages (compare what has been stated regarding cysts of the liver).

The previously mentioned regressive metamorphosis and the malignancy occasionally observed in adenomata of the liver necessitate their extirpation as soon as practicable. A diagnosis without opening the abdominal cavity is, unfortunately, impossible. A solid tumor, small or large, sometimes of the size of a child's head, often movable, which causes subjective difficulties (pain) the existence of which dates back "several years," but which evidently grows, and a diffused enlargement of the liver which in a young or older individual may be marked—this is the usual clinical finding. The recognition is made difficult by the circumstance that the tumor adheres to the liver, is tongue-shaped or even pediculated, therefore, cannot always be definitely referred to this organ. (Successful operations have been performed by Keen, v. Bergmann, Tricomi, Grube, F. Schmidt and others. Grube's patient died 11 months later from cancer of the liver.)

The history of *carcinomata hepatis* is a comparatively recent one. It is true that literature previous to the nineteenth century contains mention of cancer of the liver (van Swieten, Morgagni, Ruysch, Stoll), but it is not separable from gumma, abscess, cirrhosis, and other hepatic affections. Bayle and Cayol (1812) are usually mentioned as the first who attempted, at least pathologico-anatomically, precise differentiation (from tubercle). The clinical differentiation between primary and secondary carcinoma of the liver is ascribed to Monneret. Prior to the beginning of the sixth decade of the last century it was believed that carcinoma of the liver was usually of primary development, or, at least, was as often primary as secondary. Bamberger (1864) was the first to state that hepatic cancer was not only rarely a primary affection, but was much more frequently secondary. Virchow proved that the liver is exceptionally fertile soil for the deposition of carcinomata from all parts of the body, and that insignificant primary tumors give rise to enormous secondary neoplasms of the liver; following this, for a brief period, the possibility of primary cancer of the liver was even denied. With increasing knowledge of the histologic structure of the liver and its epitheliomata, the view that carcinoma may actually develop from the liver cell itself has again been expressed, and, unfortunately, is abundantly confirmed by clinical experience. Hanot and Gilbert estimate that for every 8 secondary cancers of the liver there is one primary. Hansemann examined in this respect the reports of the Berlin Pathologic Institute from 1870 to 1889, strictly investigating each individual case, and found among 258 carcinomata of the liver 25 primary neoplasms of the gall-bladder, 2 primary cancers of the large biliary passages, and only 6 primary tumors of the liver (concerning 2 of which he was not positively certain). According to my experience (which is confirmed by my colleague, Eppinger), primary cancer of the liver is comparatively more frequent in Graz than in Prague or Vienna. An insight into the pathogenesis and finer structure of carcinoma of the liver has been given us by the investigations of Naunyn, Waldeyer, Schüppel, Fetzer, Perls, Rindfleisch, Kelsch-Kiener, Weigert, Sabourin, Simmonds, Hanot-Gilbert, Hansemann, and v. Kahlden, which will not be here minutely considered. The clinical differentiation between adenoma and carcinoma, in so far as may be seen from these investigations, is still impossible, a view which has been previously stated.

For a clear conception of the forms of primary cancer of the liver, not only in a pathologico-anatomical but particularly in a clinical respect, it is best to follow the division of Hanot-Gilbert. They differentiate three main types: First, the *massive cancer,* which appears alone; that is, forms but a few nodules; secondly, the *nodular cancer* in which, as is the rule in metastatic carcinoma, the nodules are more numerous; and, thirdly *cirrhosis carcinomatosa,* which designation is based on the simultaneous occurrence of an epithelial neoplasm and a diffuse cirrhotic hardening of the hepatic tissue. This last-named form included (at least to th greatest extent) the previously described primary epithelial neoplasm called multiple hepatic adenomata (Kelsch-Kiener, Simmonds, Sabourin) and the *diffuse cancerous degeneration* also called *infiltrated hepatic ca cinoma* (Schüppel). (Ziegler adds to these a fourth variety of canc consisting of multiple nodules which develop from the periportal co nective tissue and break into the neighboring hepatic tissue.) Final Orth and Hansemann describe a form which originates in the *small bilia* *passages,* and, *tree-like in its course,* spreads through the liver from Glisson's capsule. Strictly speaking, we are, at least, dealing with a neoplasm of the biliary passages, such as primary carcinoma of the hepatic duct, etc.

The very rare *massive cancer* (Gilbert), the carcinoma "*of the nature of a solitary tumor,*" develops (preferably in the right lobe) as a solitary, rapidly growing, sharply defined tumor, nearly globular in shape, occasionally of very marked dimensions, sometimes surrounded by intact hepatic tissue (almond cancer) which only late in the course causes metastases in its adjacent areas. The markedly enlarged, diffused organ (sometimes weighing 10 kilograms) retains its shape, and the surface either remains smooth or only small prominences form. The non-implicated hepatic tissue, however, is not cirrhotically indurated; numerous connective tissue branches envelop isolated nodulated portions of the secondary mass. The lymph-glands of the hilus, the gastrohepatic, the peripancreatic, the prevertebral and the mediastinal lymph-glands often show malignant infiltration. Metastases external to the liver are frequently absent; by continuity the cancer occasionally proliferates into the gall-bladder and the right kidney; the peritoneum may also be attacked. Specific emboli may find their way into the hepatic veins, and develop into nodules in the lungs. Perihepatitis and ascites are comparatively rare. The spleen is usually enlarged.

The *nodulated carcinoma* forms numerous secondary masses (occasionally one large and several smaller ones), varying in size from the head of a pin to an orange, and differing in consistence and color. The liver in these cases is often enormously enlarged, its surface becomes nodulated; umbilication is, however, rarer than in metastatic carcinoma with the same dissemination. According to the structure the nodules may resemble the so-called nodular hyperplasia, the trabecular adenoma, and true cancer formation. Distributed connective tissue proliferation in some areas, with induration of the tissue, and atrophy of the parenchyma at other points, as well as perihepatitis, is frequent. Occasionally there is right-sided pleurisy. Enlargement of the spleen is found in about one-half of the cases. The lymph-glands show the same development, likewise the metastases.

In frequency this form corresponds to *cirrhosis carcinomatosa,* in which the nodular (or massive) cancer is not only in juxtaposition with the cirrhosis, but there is a genetic connection between the carcinomatous development and sclerosis of the liver; the latter is sometimes but slight, and, under some circumstances, may naturally also be markedly diffused and the liver enlarged; besides, we find connective tissue proliferation which is uniform, mostly annular, rarely in isolated arrangement; numerous trabecular epitheliomata, that is, adenomata and carcinomata, which are situated deep in the liver tissue (upon section these are quite uniformly distributed, and are often surrounded by connective tissue capsules) or (more frequently) upon the surface of the organ, or, perhaps, protruding beyond the latter in such a way that cirrhotic granula may be found lying beside cancerous nodules. The volume of the nodules varies; they may be the size of a millet seed or lentil to that of a pea or hazel-nut. This carcinomatous development rarely penetrates into the lymph-glands, but it extends into the venous system much more frequently than any other cancerous variety; in more than one-half of the undoubted cases the secondary mass enters the large afferent and efferent veins of the liver. Secondary nodules are found in the lungs (subpleural); more rarely in the peritoneum (Douglas's sac). Perihepatitis is usually present; ascites is almost invariably added. In more than 50 per cent. of the cases the spleen is enlarged and hardened, dilatation of the subcutaneous veins of the abdomen is frequent, even severe hemorrhages from varicose dilatations of the esophagus occasionally appear (original observation). "Infiltrating cancer," which this form, at first sight, is called at the autopsy, gives the impression of being cirrhosis of the highest grade. Only by microscopic examination do we learn that the islands of cells in isolated areas which resemble resistant hepatic tissue really consist of cancerous masses.

Hemorrhages into and from the carcinomatous liver, which are the result of trauma, of erosion, or of acute congestion and venous hyperemia, are occasionally of clinical importance, especially if the cancerous tissue itself is very rich in vessels, that is, if cavernous capillary dilatations are present. The blood either enters the secondary mass and the neighboring hepatic tissue, or it flows, as I saw in a particularly severe case (potator strenuus), from a portion of the cancer perforating Glisson's capsule into the abdominal cavity. Here it is sacculated by previously formed adhesions in the bursa omentalis, in Douglas's sac, etc.; or it forms a hematoma above the liver (Rollett), or, finally (rapidly fatal), it is freely effused into the abdominal cavity, and is discharged from the peritoneal sac through the inguinal and femoral canals, then infiltrating the subcutaneous tissue.

In regard to *"recovery"* by means of *retrogressive, fatty,* or other metamorphosis which the cancerous nodules of the liver often anatomically present, and the tough, cicatricial masses which are formed thereby, a process which Oppolzer, Bochdalek, and Henoch held to be possible, scarcely anything need to-day be said.

Both sexes appear to be affected by primary cancer of the liver in equal proportions; but cirrhosis carcinomatosa is more frequent in men, presumably on account of its etiologic relation to the abuse of alcohol; this appears as an epiphenomenon to alcoholic cirrhosis. Secondary carcinoma of the liver (for obvious reasons) is more often found in women. The majority of cases occur between the 40th and 45th years; naturally, primary carcinoma of the liver also occurs in advanced age and, exceptionally, in early life, even in children. Heredity and trauma are important etiologic factors. Malaria is said to be a predisposing cause. Some authors believe there is a significant relation to cholelithiasis, since gall-stones are not rarely found besides carcinoma of the liver (Biermer).

The statement is commonly made that the clinical history of cancer of the liver presents such a varying picture that a general description is scarcely possible. My own experience leads me to agree with Hanot and Gilbert that, at least in certain stages of the course, the three main anatomical groups may be clinically separated without discussing too-far-reaching theories, although, naturally, the special diagnosis is impossible in some cases. Of massive cancer of the liver, I have collected no individual observations (Gilbert mentions 8 cases with autopsy); but I may cite 7 clinical histories of my own (including autopsies), among them 3 cases of cirrhosis carcinomatosa.

CLINICAL TYPES

1. **Massive Cancer.**—Stages of development: Dyspeptic symptoms. Sometimes pain, emaciation, increasing muscular weakness. Pallor. At the height of the disease: Tympanitic abdomen, particularly in the upper areas. Enlargement of the entire liver, and extending below the level of the navel to the anterior superior spine of the ilium. Consistence of the organ markedly firm and hard as wood. Surface remains smooth. Final stages: Enlargement (moderate) of the spleen; ascites or dilatation of the veins in the abdominal skin is rare. In some cases: Hypocholia or acholia of the feces. Jaundice usually absent. Finally, anorexia; rarely vomiting. Constipation (also sometimes diarrhea). Occasionally, and in paroxysms, exacerbation of the pain. In proportion to the hyponutrition, diminished excretion of urine. Albuminuria usually absent. Fever (100.4°–102.2° F.) seldom occurs. Course always progressive, rapid, leading to marked cachexia with anasarca. Frequently autotoxic coma. Total duration: 1 to 5, more rarely, 6 to 7 months.

2. **Nodulated Carcinoma.**—At the onset: Digestive disturbances, aversion to meat, etc., pain in the right hypochondrium. Exceptionally, early jaundice. After a few weeks, enlargement of the abdomen. At the height of the disease: The enlarged liver, under some circumstances, raises the diaphragm perceptibly, enlarges the right side of the thorax, and causes the hypochondrium and epigastrium to bulge more decidedly. In a downward direction the organ extends below the arch of the ribs, its anterior border below the navel, and at the height of the spine of the ilium it reaches the margo crenatus lienis. The surface of the liver is no longer smooth, the edge no longer sharp; these changes are caused by multiple tumors which may be small and numerous, protruding semiglobularly, hard or soft, or may be less in number, voluminous, flat, sometimes even confluent, as if composed of a single tumor. Umbilications are usually

not perceptible. Occasionally a very distinct "shadow" of the liver is seen; sometimes peritoneal friction in the hepatic region. Recognizable continuous growth of the liver under the eyes of the observer. The spleen is either normal in size or only moderately enlarged. In advanced stages of the disease, in more than one-half of the cases, there is serous or hemorrhagic ascites, the amount of fluid often remaining small, and only occasionally necessitating aspiration. Sero-fibrinous effusions occur only in secondary peritoneal carcinosis. The specific gravity of the ascitic fluid varies between 1.010 and 1.020. The amount of albumin may be from 1 to 4 per cent. Pain is almost always present, but varies greatly in degree and character (radiations to the right shoulder, attacks of marked dyspnea, similar to that in diaphragmatic pleurisy). In one-third of the cases jaundice is absent during the entire course; in another third, the icterus is moderate, occasionally only just before death, and, in the last third of the cases, marked permanent jaundice which is not subject to variation develops (by compression of numerous intrahepatic biliary passages, or of the common gall-duct by cancerous hilus ganglia, or by large intumescent gastrohepatic and peripancreatic lymph-glands). Constipation. During the latter period there is vomiting, especially when there is rapid growth of the tumor. Acholia in consequence of hepatargy is rare. Profound anemia, cardiac asthenia, variable temperature, usually normal, but toward the end with moderate fever, may occur. Long-continued high fever is observed only in isolated cases when the tumor enlarges very rapidly; sometimes it may be combined with chills, it may be remittent, or even continuous. Total duration: A month to a year; the average is 3 to 6 months. The younger the individual, the more rapid, as a rule, the course. Death may take place in coma; more frequently it occurs with the symptoms of sheer exhaustion. Besides the ordinary form just depicted, Hanot and Gilbert describe quite a number of special "varieties." I concur with them only so far as their observations accord with my own experience and that of other authors, and must reiterate that the clinical symptoms of nodular cancer are, in fact, but little less characteristic and uniform than those of other types of carcinoma hepatis. Fever is occasionally prominent in the clinical picture, or the cancer may betray itself by the predominance of a cachexia, while, in other cases, the body retains its fulness for some time. Carcinoma may, during long periods of its course, produce no other functional symptoms than paroxysms of intense pain; in other cases, continuous vomiting is a prominent feature. In conclusion, permanent jaundice may play a leading rôle.

3. **Cirrhosis Carcinomatosa.**—Symptoms at the onset: Dyspeptic disturbances (loss of appetite, sense of weight in the stomach, flatulence, eructations, vomiting). Indefinite pain, loss of strength, emaciation, anemia. Frequent epistaxis. Early enlargement of the abdomen. At the height of the disease: Cachexia, ascites, often increasing so rapidly that

aspiration becomes necessary. Dilated veins of the abdominal skin. The liver may be enlarged, and increase under the eyes of the observer; or, it cannot be palpated, and decreases in size. If still palpable, the surface appears coarse, nodular, and occasionally granular. The spleen is often enlarged (but only moderately), even palpable; sometimes, however, it is normal. Jaundice is found in all the well-marked cases; it occurs early or sometimes even late. There may be constipation, or, on the other hand, diarrhea; toward the end the feces become acholic. Hematemesis and melena appear. Fever plays no rôle; toward the end there is edema. The total duration of the disease is 1 to 16, upon an average, 4 to 5, months. After anasarca has appeared death occurs in coma and from extreme exhaustion.

As will be noted, in the *development* of the three anatomical types of primary cancer of the liver, the same constitutional disturbance, disorders of digestion, as well as very similar, but only slightly characteristic, pain, and the wretched appearance of the patient are common. Absence of jaundice is the rule. Massive cancer, and cirrhosis carcinomatosa with a relatively small liver, often exist for some time without causing local signs. Symptoms such as those of Bogdan (flaky, wine-red discoloration of the cheeks, and varicosities of the superficial capillaries of the same region occurring in gastric and uterine carcinomata), or that of Mettenheimer (marked emaciation of the epigastric region with an especial distortion of the navel, "navel-fold," in cancer of the abdomen) is scarcely of decisive import. The tenacity and, still more, the visibly rapid progression of the previously mentioned symptoms would incline us to the first suspicion rather than to the view of a mild gastric or intestinal affection. But a careful study of the clinical reports of other authors and my own experience teach me to beware of the danger that lurks in the latency of the disease and the absence of objective symptoms referable to the diseased organ, which, as a rule, continue until it is too late for the physician to accomplish anything effectual. Of the value in such cases of a possible diagnostic exploratory laparotomy we shall speak later. In the transitional period, the main stress is to be laid upon the parallelism of an increasingly rapid loss in weight, and a rapid enlargement of the entire liver. Quickly as a carcinomatous liver grows, as a rule only certain forms of congestion develop, and these are usually not difficult to exclude. There is scarcely a doubt of the fact that when a palpable tumor of the liver can be determined, it is too late for effectual operative interference. Two initial symptoms upon which much stress is often laid are hyperleukocytosis and the decreased amount of urea in the urine; the first is of minor importance, and the second of scarcely any differentio-diagnostic value. In my experience, leukocytosis (and especially certain forms of digestive leukocytosis) favors cancer; I have reason, however, to doubt that this actually occurs early. The symptom, too, is variable. In my cases, the number of leukocytes to the cubic millimeter varies between 6,500–18,000; but my experience compels me to differ from the view that urea and chlorin are invariably decreased in the urine in the early stages of cancer of the liver. The amount of urine and nitrogen in carcinoma hepatis depends, in the main, upon the degree of inanition existing, and a rapidly increasing ascites may influence the excretion of water. In the earlier stages, and even at the height of the disease, I found medium amounts of urea, and it was possible even to bring some patients temporarily into N-equilibrium! Alimentary glycosuria plays no diagnostic rôle, and acholia is not an actual early symptom.

DIAGNOSIS

At the height of the disease we may adhere, in a diagnostic respect, to the previously described types. In practice, as soon as a well-developed cachexia is observed, or a palpable tumor of the liver appears combined with symptoms of compression in the region of the portal vein and in the biliary passages, a special differentiation is only of importance as an indication that operation is unnecessary. All patients succumb soon or late, and, therefore, suffer to about the same extent. Ascites is a frequent hindrance to the recognition of the affection in its advanced stages. Special diagnostic consideration of compression (simultaneous) of the biliary passages and the portal veins has already been referred to; all carcinomatous tumors of the liver are characterized by the great increase in volume which is not significant in any other affection of the liver. The cases of soft cancer of the liver, in which the sensation through the abdominal wall simulates fluctuation, are rare exceptions. In a differentio-diagnostic respect, besides massive cancer, we must consider the following: Melanomata (which usually also cause enlargement of the liver with a smooth surface, but in most cases are secondary to neoplasms of the eye or skin, and give rise to melanuria), Hanot's hypertrophic cirrhosis (having a slower course without pain, cachexia and marked anemia, hypercholia, that is, so-called poussées, jaundice, hard splenic tumor), Hutinel-Sabourin's cirrhosis adiposa hypertrophica, certain forms of congestion and enlargement of the liver in diabetes, malaria, syphilis, leukemia, pseudo-leukemia, amyloidosis and cholelithiasis. Many forms of nodular cancer so completely fill the abdominal cavity that it becomes difficult to determine the point of origin. Perplexity in diagnosis may also arise when the cancerous nodules coalesce and form a few larger structures or a tumor. Confusion with secondary hepatic cancer is a frequent occurrence. When a tumor of the liver is present we should never fail carefully to examine the entire body for a primary neoplasm (by palpation of the stomach, digital examination of the rectum, of the vagina, etc.). In opposition to Leube's view, I must state that the determination of the acidity of the digestin stomach permits no decisive conclusion; even in unquestioned primary cancer of the liver, free hydrochloric acid may be absent. Pulmonary and pleural cancers, as previously indicated, occur secondarily somewhat more frequently in the course of cancer of the liver; similarly mediastinal and peritoneal tumors, but rarely growths in the abdominal wall (umbilicus) and small tumors. The nodules of primary carcinoma are generally harder than those of secondary cancer. The umbilication is frequently unrecognizable. In secondary cancer of the liver there may be no enlargement of the organ, and the tumor of primary cancer of the gall-bladder may, in most cases, be diagnosticated with certainty according to criteria which have been enumerated. Exceptionally, carcinoma ventriculi causes distributed tumor formation which resembles liver nodules, and certain neoplasms of the omentum may simulate the form of the liver. Cancer of the right kidney has been repeatedly confounded with carcinoma of the liver. The nodular surface of nodular cancer is common also to syphilitic liver, to multilocular echinococcus, and to liver cysts of non-parasitic origin. We differentiate by the slighter resistance of the protuberances, the slow development and the late cachexia, besides the history and other symptoms (for example, the condition of the spleen). Syphilomata may occasionally appear as isolated tumors of the size of a fist (more or less painful) below the right arch of the ribs in the diffusely enlarged liver, which is of normal consistency. Such gummata have been mistaken for cancer and extirpated. But, inversely, syphilomata have been assumed, and mercury has been administered until, on exploratory laparotomy being later performed, an inoperable soft cancer has been revealed. Even confusion with chronic abscess of the liver has occurred. In cirrhosis carcinomatosa the symptoms vary according to the degree of cirrhosis present, this depending upon the situation, the number and the size of the cancer nodules, that is, the infiltrated carcinoma mass. Correspondingly the liver is sometimes large, or very large, and confusion with nodulated cancer or secondary hepatic carcinoma is possible; under these circumstances marked ascites is a diagnostic warning. In another series of cases with but slightly enlarged liver which slowly decreases under observation, especially if alcohol may be regarded as the cause, we are inclined to make a diagnosis of cirrhosis. As is well known, there are also forms of the latter disease which run a subacute course, in which marked parenchymatous degeneration of the cellular elements of the liver plays an important rôle. Generally, however, it is cirrhosis carcinomatosa in which the rapid development and severe nutritive disturbances are peculiar, and which soon terminates in death. Finally, there are cases in which the right-sided pleural exudate early forms and appears to explain the entire clinical picture.

In extremely rare cases only does the enlargement of superficial lymph-glands assist us in the diagnosis of cancer of the liver. The jugular gland, especially, plays no rôle. Enlargement of the inguinal glands is somewhat more frequent.

Except from the stroma, *secondary cancer of the liver* originates exclusively from cellular elements which have been detached from an extrahepatic carcinomatous neoplasm, and by the lymphatic, arterial, and venous channels have reached the liver to which they adhere, then proliferate in the capillaries. There is scarcely an epithelial tissue from which the liver may not thus be secondarily affected. Obviously a special tendency to infection of the liver is found in carcinomata of those abdominal organs which are closely related to the portal vein (stomach, rectum, intestine, pancreas, biliary passages). I am in possession of accurate histories and autopsy reports of 27 cases of secondary cancer of the liver. The primary neoplasm was 15 times seated in the stomach (in 2 of these cases only the left lobe showed circumscribed implication), 4 times in the gall-bladder, in 4 cases in the rectum, in 2 in the kidney, and in 2 in the esophagus. Hepatic metastases are, however, also found in primary cancer of the testicles, of the seminal vesicles, of the prostate gland, of the uterus, of the ovary, of the breasts; in the lungs they are exceedingly rare. The neoplasms may occur secondarily outside of the actual portal vein area (due to other metastases), having reached the portal vein system prior to their deposition in the liver. The secondary cancer of the liver possesses an extraordinary tendency to form multiple nodules, which, in regard to their number, volume, consistence and shape, do not differ from cancer nodularis. As a rule, the liver shows very diffuse enlargement, attaining a weight of from 5 to 10 kilograms); its surface becomes nodular. Exceptionally the secondary growth is limited to the left lobe of the organ; more rarely does diffuse infiltration occur (Litten). The conspicuous tendency to retrogressive metamorphosis, in contrast to primary carcinoma, is usually looked upon as the cause of the marked umbilication. Breaking down of the cancer may also lead to the formation of cysts, to hemorrhage, even to rupture and bleeding into the peritoneal cavity.

TREATMENT

The limits of operabilitas hepatis quoad resectionem, as it is called by Langenbuch, as well as the results of experimental investigations and of operations already performed in man (and even excluding from consideration the great danger of hemorrhage), are such that the justification for, and the technical possibility of, the removal of malignant tumors of the liver is out of the question. The animal experiments of Ponfick, which have become celebrated, and later those of Meister, Kusnezow, Penski and others, are decisive indications for this proceeding in surgery of the liver, in that they have absolutely proven that the removal of even a relatively large portion of the liver may be borne without serious damage.

Any one who has carefully examined a number of carcinomata and sarcomata of the liver must admit that the reason why tumors of the liver are rarely removed is not the impossibility of the operation, but the difficulty of early recognizing the indication for the operation, and its consequent poor results explain why surgery, which is constantly advancing into wider domains, unfortunately cannot completely master this organ. Occasionally it may occur that a gastric, intestinal or gall-bladder cancer may be removed, also simultaneously a portion of the liver, which is exclusively the seat of a beginning secondary carcinomatous development. In all other cases, metastatic carcinoma or sarcoma will only be an absolute contraindication to operative procedure. At most in a very small fraction of the rare primary malignant neoplasms of the liver is there any indication for surgical treatment. Certain types of carcinoma, for example cirrhosis carcinomatosa, which is observed with comparative frequency, and also most cases of nodular cancer, are not suitable for this kind of treatment. The peculiar structure of the liver which greatly favors the rapid distribution of an originally circumscribed affection to wide areas of the organs, the multiplicity of the malignant neoplasms which soon become obvious, the difficulties of an early diagnosis which have been previously described, the history which for the most part only points to a disease of the intestinal system, are all observed too late to be absolute indications for operative treatment directed to the liver. Many surgeons, therefore, hold that these early symptoms above described, which with more or less positiveness point to a serious abdominal disease, indicate an exploratory laparotomy. In this manner a tumor of moderate size, from that of a hen's egg to an apple, and favorably situated, might be removed; personally I know of nothing that contraindicates an exploratory laparotomy. If we consider, however, how often on such vague signs this operation would be resorted to in practice, and how frequently, from obvious diagnostic errors, it would be practised unnecessarily, it is easy to understand that the carrying out of such a proposition would meet with objections on the part of the patient and his relatives and would even be objected to by those physicians who themselves have committed a few errors. Therefore, many favorable circumstances must be combined to enable us to perform resection of the liver to the benefit of the affected patients. To Hochenegg belongs the honor of being the first successfully to remove a cancer of the liver from its point of origin in the gall-bladder. A case of Lücke's is very instructive as a guide to the decision for the operation. This case was published in 1891, and, on account of the successful removal of the left lobe of the liver, it was generally looked upon as an example of a pure primary cancer of the liver until a more recent publication of Madelung (1898) showed that it was only a syphiloma.

ECHINOCOCCUS OF THE LIVER[1]

BY E. STADELMANN, BERLIN

CLINICALLY and anatomically we may differentiate two varieties of echinococcus in man: 1. Cystic echinococcus; 2. Multilocular or alveolar echinococcus. We know that the echinococcus cysticus may develop (Siebold, Küchenmeister, Leuckart) whenever the ova of the tenia echinococcus which lives in the intestinal canal of the dog, the wolf, the jackal, etc., are found in the gastrointestinal canal of man. The embryo, which is set free by the digestion of its chitin covering, burrows through the intestinal wall, reaches a branch of the portal vein, and enters the liver with the circulation; here it lodges and is transformed into an echinococcus cyst. Naturally, the echinococcus develops not only in the liver but also in many other organs (lungs, spleen, brain, kidneys, etc.), provided it can find ingress by means of the circulation or the lymph channels. In its early existence the echinococcus cyst develops but slowly, first to the size of a tubercle, later to that of a pea, but it is a firm nodule with a thick wall which is composed internally of muscle and vessels (parenchymatous layer), and externally of a tough, lamellar layer of tissue (cuticula). In the affected organ, and around the cyst which is filled with a clear, non-albuminous fluid, a tough connective tissue capsule forms which may be easily peeled from the nodule, and in which are many vessels by means of which the echinococcus is nourished. After four to six months the growing vessel attains the size of a walnut, and in its interior (endogenous), very rarely externally (exogenous), daughter-cysts now begin to develop (secondary hydatid cysts), even granddaughter-cysts (tertiary hydatid cysts) which are partly sterile, and partly contain the heads (scolices) of the echinococci. These heads have the characteristic shape, and are supplied with suctorial discs and a crown of hooklets. Usually the daughter-cysts become detached from the inner parenchymatous wall, and float free in the fluid, so that in such a mother-cyst hundreds of daughter-cysts may be found.

The development of the multilocular echinococcus is different. Virchow first recognized this as a parasite; it forms a coarse tumor composed of vesicles the size of a pea. In contrast with the cystic echinococcus, its growth, therefore, takes place by exogenous proliferation in that one echinococcus vesicle attaches itself to another. It betrays the same nature as malignant neoplasms, and not only attacks the parenchyma but also the blood-vessels, such as the portal vein, the hepatic veins, the hepatic arteries and the lymph-vessels. Constriction may take place, and, as in malignant neoplasms, give rise to metastases. It is a mooted question whether multilocular echinococcus is identical with cystic echinococcus, or whether we are dealing with two different varieties of teniæ. A number of factors favor the latter view; for instance, the circumstance that echinococcus multilocularis occurs in definite localities (South Germany, Austria, and parts of Russia) while in other regions (Iceland, Mecklenburg, and Pomerania) the echinococcus cysticus preponderates, and also that some authors by feeding experiments with the echinococcus multilocularis succeeded in producing this parasite experimentally; this statement, however, is denied by other authorities. Finally, the scolices of these forms are said to differ from each other, particularly in the shape of their hooklets. No matter how this may be, the question whether we are dealing with two different varieties of echinococci or whether the same echinococcus produces two different forms of the disease, must be looked upon as still in dispute.

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[1] See also the article " Echinococcus Disease " by Prof. E. Peiper in the volume on "Diseases of Metabolism and of the Blood; Animal Parasites; Toxicology," page 526.

A. ECHINOCOCCUS CYSTICUS

PATHOLOGICAL ANATOMY

The origin and development of the vesicles, as well as the configuration of the walls of the cyst, have already been described. Besides daughter-cysts, the vesicles contain a clear, almost invariably non-albuminous fluid with a specific gravity of from 1.009 to 1.015, and varieties of sugar (grape-sugar, inosite), succinic acid and salts (principally sodium chlorid). According to the investigations of different authors (Mourson and Schlagdenhauffen, Brieger), toxic substances are also present which may be assumed to be ptomains, since, when the contents of the cyst are injected into animals, they produce collapse and death. Echinococci are usually found in the right lobe of the liver. The vesicles may develop in any region of the liver, upon the upper surface (the convex), upon the lower surface (the concave), centrally as well as subphrenically. The symptoms of displacement of other organs, as well as the extent of the growth, frequently also depend upon the seat of the echinococcus, and this again produces the clinical symptoms as well as the diagnostic determination of the pathologic process. We would digress too far if we entered upon the individual points which are self-evident. It need only be stated that the tumor may extend to the crest of the ilium, may force the diaphragm up toward the pleural cavity, may sometimes be found deep in the pericompanied by pain. After rupture, the cyst rapidly collapses, the feces become watery, and sometimes contain blood, hydatids, or their remains. The lesion of communication may close or it may persist. In the latter case fecal contents find their way into the liver, and there develop a fecal abscess.

Perforation into the peritoneal cavity results when there have been no adhesions to neighboring organs, as is so frequently the case, but contusions, trauma, sudden violent exertion, force or pressure, etc., may be the cause. The results of rupture are severe shock, syncope, great abdominal pain, mental disquietude, the sense of impending death, or even sudden dissolution. The patients feel as though a part of the abdomen had been torn; they are pale, anxious, and distressed. Urticaria frequently appears in consequence of the toxins which have developed in the fluid. If the patients do not succumb to the rupture the hydatids may still further develop in the abdominal cavity, and if the generators of infection contained in the fluid of the cyst are expelled into the belly, peritonitis follows. This process is much more favorable if adhesions to the peritoneum have previously formed, and if the cyst ruptures into an encapsulated portion of the abdominal cavity, so that diffuse peritonitis does not result.

Rupture into the urinary passages is very rare, but may lead to renal colic and hydronephrosis when the hydatids are caught in the ureter. When they are voided by the urinary channels, we find in the urine cysticerci vesicles which are occasionally bile-stained.

Rupture into the pleural cavity results in cysts in the subphrenic space. According to the composition of the exudate, we then have the signs of serous or purulent pleurisy. By exploratory puncture we obtain some of this fluid, and, microscopically, we are sometimes able to make a correct diagnosis by demonstrating hooklets or portions of the membrane. The slight amount of albumin, the clearness of the serous fluid, or its bile-stained appearance, may awaken our suspicions that it comes from an echinococcus cyst. The rupture of the sac gives the patient a sensation as though something within the chest had been torn, then follow a feeling of anxiety, dyspnea, or even collapse. The pulse becomes small and frequent, and with these symptoms death may soon occur. If the contents of the cyst are purulent, the well-known symptoms of empyema with pain and fever are produced. The empyema, if not recognized in time and treated by operation, may rupture externally or into the lung. In th latter case, occasionally with severe paroxysms of coughing, great number of hydatid vesicles are coughed up, and complete discharge of the con tents of the cyst as well as cure results. In other cases a pulmonar abscess forms; pneumothorax from the rupture of an exudate into a bron chus, and even direct rupture of the contents of the cyst into the lun without the development of an empyema such as has been described or pleurisy, have been reported. If rupture into the bronchi or the trache occurs too rapidly in consequence of the great mass of fluid and hydatid cysts expelled, the patient may be suffocated. Large cavities may develop and produce corresponding phenomena. Upon rupture of an echinococcus cyst into the lung the sputum is frequently bile-stained.

Rupture into the pericardium is most infrequent, and probably in all cases soon causes death by paralysis of the heart.

Rupture into the portal vein produces multiple abscesses in its various branches.

Rupture into the vena cava permits the entrance of hydatids into the circulation, thence to find their way into the right heart, and later into the pulmonary veins, producing pulmonary emboli with all the symptoms of a hemorrhagic infarct or even a pulmonary abscess.

Suppurating cysts may also rupture externally through the abdominal wall, the same as hepatic abscesses, unless they are early recognized.

DIAGNOSIS AND TREATMENT

Small echinococcus cysts cannot be recognized. They are, however, occasional autopsy findings. The recognition of large cysts is mainly due to their position, but even then they are frequently overlooked, especially if they produce no symptoms and their position is concealed. If we discover a tumor which we suspect to be an echinococcus, this diagnosis rather than that of other tumors, particularly malignant ones, is favored by the slighter pain, the round, smooth surface, the mild disturbance of the general condition, the slow growth, the tense, elastic consistence, perhaps also by a positive hydatid thrill. If remittent or intermittent fever coexist, the differential diagnosis between abscess of the liver and a suppurating echinococcus cyst may be impossible without surgical intervention.

In the diagnosis of a subphrenic cyst the X-rays, as well as the observation of Litten's diaphragm phenomenon, will be of value. In a pleural exudate the diaphragm is forced down, in a subphrenic echinococcus, on the other hand, it is forced upward, while the remaining clinical signs in these pathologic conditions differ but little from each other.

Cysts of the spleen, situated near the left lobe of the liver, may be mistaken for echinococci, also those of the pancreas and even of the ovary, as well as dropsy of the gall-bladder and a right-sided hydronephrosis.

In many cases exploratory puncture with the subsequent chemical and microscopical investigation of the cyst contents which have been obtained may clear up and decide this question. This is, therefore, an important diagnostic aid which may be employed without danger in case of a puncture of the pleural cavity, provided antiseptic precautions are taken. On the other hand, puncture of the abdomen is decidedly hazardous. Even thin cannulæ will produce holes in the tense cyst wall from which other contents may readily ooze, and, particularly if they are infectious, lead to general peritonitis. Therefore I earnestly advise that puncture be performed only upon the operating table, and when operation is to follow immediately; in the interests of the patient it is well to leave a definite diagnosis to the surgeon. It is certainly fortunate for the patient if the physician decline the triumph of making the decision, and work hand in hand with the surgeon. He may do this all the more conscientiously since, if echinococcus be present, the only possible treatment is by operation. The cyst must be opened, and, if it prove to be an echinococcus cyst, the contents must be evacuated. How this is to be done, and which method of procedure is the best, is difficult to decide, as the situation of the cyst in the individual cases may necessitate different methods. Neither can it here be positively stated whether the single or the double method will give better results. At all events, it should be the earnest endeavor of every surgeon to avoid in every possible way the infection of the neighboring organs by the fluid evacuated from the cyst. After the parasite has died some cysts heal by calcification.

Drug treatment is wholly ineffectual, and it is advisable not to attempt it.

B. ECHINOCOCCUS MULTILOCULARIS SEU ALVEOLARIS[1]

The question as to the manner in which multilocular echinococcus differs from cystic echinococcus has been discussed, as well as the nature of its development. The typical difference is that multilocular echinococcus forms in the cavities of the liver (bile ducts, blood-vessels, lymph-channels) and extends by gemmation, destruction going on in the interior of the colony, and thus cavities differing in size are formed, also that its action in the liver resembles that of malignant neoplasms since it destroys the actual hepatic tissue and at the same time causes an extensive connective tissue proliferation. In consequence of this mode of development, the appearance of the liver in echinococcus multilocularis may vary greatly.

If the tumor is situated in the interior of the organ, the form of the liver is not necessarily altered and the disease process is revealed only by section. In other cases, nodular protuberances of varying size are found upon the surface, and between these are connective tissue retractions; occasionally distinct echinococcus cysts are recognizable in the nodules. The consistence of the liver is sometimes very dense owing to a large deposit of chalk frequently found in the connective tissue. In old echinococcus cavities in the interior this condition is found as the consequence of decomposition which has taken place. The liver is usually, and sometimes greatly, enlarged, cases of echinococcus multilocularis having been described in which two liters of fluid were evacuated from the cavities. Vicarious hypertrophy of the liver substance, chiefly of the left lobe (the echinococcus usually develops in the right lobe), has been reported.

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[1] For the literature compare the following works: *Hoppe-Seyler* in "Die Krankheiten der Leber von Quincke und Hoppe-Seyler"; *Nothnagel's specielle Pathologie und Therapie*, XVIII, Theil 1; *Vierordt, Berliner Klinik*, Heft 28, and the monograph upon "Multilocular Echinococcus" published in Freiburg in 1886; *Posselt, Deutsches Archiv für klin. Medicin*, LIX.

The small tumors are white to yellowish green in color, with a distinct line of demarcation from the surrounding, dark, hepatic tissue.

Section of the tissue produces a grating sound, the cut surfaces reveal numerous apertures (alveoli), hence the tissue resembles a honeycomb, and the echinococcus has been designated "alveolar." These alveoli are of different sizes, ranging from that of a pin's head to that of a pea, a walnut, an apple, a child's head, etc., and between them the connective tissue is increased. As these cavities coalesce a peculiar picture may develop; the consistence of the contents varies from that of a gelatinous material to a tough, dry, friable, even calcareous mass which is usually a bile-stained yellow. In consequence of the great destruction going on, enormous cavities may occasionally form; these are partly filled with fluid, partly with compact masses permeated by connective tissue striæ which represent the remains of large blood-vessels and bile channels. The contents of the cavities often have a thready, cream-like, gelatinous, more or less bilious or even brownish appearance, and, according to microscopical and chemical investigation, they are composed of mucus, calcium, cholesterin, remains of blood, bilirubin, hematoidin, remains of echinococci, hepatic tissue, albumin, biliary coloring matter, bile acids and fat.

Macroscopically and microscopically the hepatic tissue is greatly altered. The bile ducts are sometimes dilated by the tumors growing within them, at other times are compressed by these growths. The common bile duct may be occluded from compression; the same is true of the cystic and hepatic ducts. This produces various other anomalies such as absolute occlusion of bile with severe jaundice, dropsy of the gall-bladder, etc.

In consequence of compression of the portal vein, stasis arises with enlargement of the spleen, ascites, etc. The lymph tracts are also frequently implicated by tumors, and consequently have a peculiar appearance; they are filled with echinococci and resemble a pearl necklace or a rosary. This latter condition is even observed in the lymph-glands at the porta hepatis. Metastases of echinococci are also found in the lungs, the spleen, the mediastinal glands, the pericardium, the peritoneum, the pleura, and even in the endocardium. Besides the already described contents of the individual cavities the microscope reveals that, surrounding these tissues, a structureless membrane is first formed which belongs to the echinococcus itself and is usually distinctly layered, and subsequently an extensive connective tissue proliferation which may be regarded as a reaction of the liver to the foreign body that has encroached upon its substance. Upon the inner surface of the cavity there is also a parenchymatous layer. Hooklets and scolices are frequently absent from the primary tumor, and this coincides with the existing tendency to death and destruction of the parasite; they are, however, present in recent growths and in metastases. The membrane of the parasite itself, which corresponds to the cuticula of the cystic echinococcus, naturally consists largely of chitin.

SYMPTOMS AND DIAGNOSIS

Alveolar echinococcus runs its course for a long time without symptoms, then pressure signs, a sensation of tension, pains in the epigastrium, loss of appetite, nausea, etc., appear—symptoms which are by no means typical. Fever and even jaundice (pressure symptom) may be present. The patient's sufferings increase, and he begins to emaciate. Objectively we find a more or less enlarged liver of increased consistence, and if the echinococcus tumor be susceptible to palpation hard, nodular masses are felt. Occasionally we obtain distinct fluctuation even with a very large, superficially situated, decomposing echinococcus cyst. In other cases in which there is a hard, tough tumor of the liver, hypertrophic hepatic cirrhosis is simulated.

Sometimes the symptoms of jaundice due to compression of the common bile duct may be particularly prominent and combined with all the familiar manifestations of icterus gravis. In other cases, the signs are those of portal vein stasis with ascites, splenic tumor, etc., in consequence of the compression and constriction of this vessel which dominates the clinical picture, as well as the familiar gastrointestinal symptoms which are so frequent in portal vein stasis, such as constipation, diarrhea, loss of appetite, etc. Death results from increasing emaciation and exhaustion, edema, and cachexia, usually after a very long duration of the disease.

The *diagnosis* can rarely be made with anything approaching positiveness. In the differential diagnosis hypertrophic hepatic cirrhosis and cancer of the liver will usually come into question, according to the symptoms of the individual case. Age, the environment of the patient, the examination of the gastric juice, fever, and enlargement of the spleen will usually protect us from error in the differentiation from carcinoma. When we suspect the existence of a cavity in the liver, exploratory puncture and the subsequent examination of the contents of the cyst thus obtained will lead us to a definite opinion. Of course, in echinococcus alveolaris the nature of the contents of the cavity varies according to its seat. Sometime its constituents are biliary, such as biliary coloring matter, bile acids and cholesterin; at other times fatty, if communication of the tumor with th lymph tracts has occurred; and in still others, upon communication wit the blood-vessels, blood may be present.

In contrast with the fluid contained in the cavities of cystic echinococcus, the echinococcus alveolaris contains extremely large quantities o albumin.

When we suspect echinococcus alveolaris, exploratory laparotomy and the examination of an excised portion of the tumor is clearly indicated.

Amyloid liver and hepatic abscess, as well as cystic echinococcus, may all be considered in the differential diagnosis. The first two are easily excluded. In the last, the result of the examination of the fluid obtained by exploratory puncture is decisive.

PROGNOSIS AND TREATMENT

The disease is nearly always fatal. We may, however, hope that with increasing knowledge of the malady and a timely diagnosis, proper treatment, particularly early operative measures, may be instituted. These should consist of extirpation of the tumor and resection of the affected portion of the liver, but they can only lead to favorable results when the operation is performed in the initial stages of the affection. The possibility of this is demonstrated by facts, since a cure of the disease in this way has certainly been effected (Bruns[1]).

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[1] *Bruns' Beiträge zur klin. Chirurgie,* XVII.

GALL-STONES

By E. NEUSSER, Vienna

Cholelithiasis is a border-land for the physician and surgeon. Both are familiar with its symptom-complex, and must work hand in hand to discover the etiology, to make the diagnosis and prognosis certain, and to formulate its therapeutic indications.

Many results in this realm we owe to the physician, many to the surgeon, who actually makes autopsies *in vivo*. But, in spite of numerous researches and statistics from both sides, they have not completely elucidated even the most vital questions in cholelithiasis, such as the question of jaundice, nor even that of the mechanism of gall-stone colic.

SYMPTOMATOLOGY

The etiology of gall-stone formation, the important factor which to both would be explanatory, and would also decide the treatment, is still obscure: here aseptic inflammation, there bacterial infection; upon the one side confidence in sterile bile, upon the other the specter of the bacterium coli, the exaggerated fear of all possible anatomical complications, and the old belief in the efficacy of a mineral spring cure.

It cannot be denied that, at the present time, the surgeon's word carries the weight of authority, and already almost identifies an early operation with a radical cure. Yet he may, any day, be surprised by the discovery of an anti-bacterial serum which will render *gall-stones inert*, and the operation for radical extirpation will meet the same fate as did tracheotomy in true diphtheria.

Until this fortunate epoch arrive, the clinician, in justice to both parties, must limit himself in the given case to protecting the patient from the two extremes, eagerness to operate and failure to recognize the right time for operation.

The usual symptoms of cholelithiasis are pain in the right hypochondrium which radiates to the back and the region of the shoulder, which either sets in suddenly or rapidly increases in severity, also eructations and vomiting, chilliness or well-developed chills, sensitiveness to pressure in the region of the gall-bladder and succeeding jaundice. In the overwhelming majority of cases these symptoms will justify a diagnosis of cholelithiasis; nevertheless, there are also cases in which the same symptom-complex, in the same sequence, is present, and to our surprise, instead of finding the expected gall-stones in the feces, the passage of echinococcus cysts completely changes our diagnosis. Even from this one example it is obvious that typical cholelithiasis may cause diagnostic difficulty. The situation becomes more complex when we deal with an atypical form of this polymorphic disease setting in with strange symptoms, which may appear under the mask of a meningitis, an endocarditis, a pulmonary abscess, cancer of the stomach or intestinal obstruction.

The statistics of errors in the diagnosis of this affection disclose so many that it is evident we can only avoid confusion by the most careful investigation of individual symptoms. This necessitates the examination of the patient from head to foot, with the aid of all our modern methods; for there is obvious danger that a symptom which we have carelessly overlooked upon superficial investigation might have been decisive in diagnosis.

The most conspicuous symptom which, from the first moment of a cursory inspection, forces itself upon the attention of the investigator is *jaundice*.

Its diagnostic importance *clears up* the cases which, for a time, have been regarded as ulcer of the stomach, as cardialgia, intestinal colic, or wandering kidney; in these the appearance of jaundice alone reveals the true nature of the malady.

Jaundice in cholelithiasis is either the direct consequence of gall-stones or enlargement of the lymph-glands at the porta hepatis, or it arises independently of these by an accompanying substantive catarrh of the mucous membrane, by neoplasms of the stomach or of the duodenum, by tumors of the pancreas, or by other tumors adjacent to the liver, for example, hydronephrosis.

Jaundice is usually and most readily explained by biliary stasis due to obstruction of the choledochus or hepatic duct by stone, therefore, as Riedel calls it, *pure lithogenous icterus*. The intensity of this jaundice depends chiefly upon the size of the stones, partly also upon the degree of distention of the biliary passages from a preceding passage of stone, and partly also to the position of the stone in a possible diverticulum of the canal. If the biliary passages are dilated at the point where the stone is impacted, or if there is a groove in the stone which permits the bile to flow off unhindered, then, even with very large stones in the common bile duct, the jaundice may be slight or entirely absent; cases such as this were familiar to Morgagni.

With an obstruction by stone in the common duct, icterus may also be absent if an accessory ductus choledochus exists by which the bile may flow into the intestine, a structural anomaly which was observed by Fallopius and Vater. Frank describes a case in which the common duct bifurcated into two branches, one of which emptied into the jejunum and the other into the colon.

radiation of pains to the shoulder and chills. In the last attack the patient was permanently jaundiced.

After the appearance of the jaundice the colic did not recur. The icterus, however, remained, and showed the peculiarity that in the winter, especially when the weather was very cold, it was particularly intense, while in the summer it was less noticeable. Appetite and regularity of the bowels were undisturbed. The patient had no symptoms except continuous lassitude. An operation was performed in Albert's Clinic, and showed a stone situated in the diverticulum of the cystic duct compressing the common duct. As with chronic although varying jaundice we usually diagnosticate a lithogenous obstruction of the common duct, the history in this case is notable, particularly the increase of the jaundice in winter and its decrease in summer. This can only be explained by the hypothesis that the patient took more fluid in summer, and, by increasing the biliar secretion, the immobility of the choledochus was overcome. This observa tion is all the more interesting since, according to Hermann, jaundic due to occlusion of the ductus choledochus increases during a miner spring treatment at Carlsbad.

In cholelithiasis intrahepatica the jaundice may also depend upon tl concentration of bile. It may be entirely absent if the bile is a thin flui and, on the other hand, may be intense with an inspissated, thick, sanc pappy state of the bile (Leichtenstern).

In regard to the development of jaundice in cholelithiasis and, in fa jaundice in general, those cases are of special significance in which flow of bile is from the biliary fistula which has been produced by operati and, in spite of which, jaundice continues when no mechanical obstruct can be demonstrated in the biliary passages.

Inflammatory swelling of the cystic duct was therefore not pres consequently there was no inflammatory jaundice. It can hardly be plained in any other manner than by the view of a *direct lesion of liver cells*; whether, in Liebermeister's sense, as a diffusive jaundice as Minkowski and E. Pick believe, due to a perverted secretion of liver cells, cannot be decided at present.

These attempts at explanation coincide with the results of the in gations of Browicz, who refers all forms of jaundice to an incr function of the liver cells, which, stimulated by various influences, up excessive quantities of nutritive and functionating material, even l globin, and transform them into bile.

The circumstance that jaundice sometimes develops to its fulle tensity even a few hours after the onset of the gall-stone colic. experimental jaundice after tying the common duct requires at le hours, and usually 2 to 3 days or even longer, led Pick, in the year contrary to current opinion, to declare that jaundice in gall-ston was not obstructive jaundice, but was reflex, due to the nerves of se of the liver (*nervous paracholia*), a reflex conveyed from the sensory nerves of the gall-bladder to the nerves of secretion.

The rapidity with which jaundice develops after the beginning of the attack may also be explained by a mechanical theory, as, in fact, is done by Gerhardt in his lecture upon icterus duodenalis in which he says: "In gall-stone colic the bile in the biliary passages is not only under the pressure of secretion, but under that of the pathologically contracted gall-bladder; therefore, it passes very rapidly into the hepatic veins; 6 hours after the beginning of the attack, traces may be found, and, after 12 hours, there is a well-developed form of jaundice." These contractions of the gall-bladder, however, can only be decisive for the development of jaundice when the cystic duct is permeable, therefore in ordinary cholelithiasis with the passage of the stone through the cystic duct; while in the irregular forms of cholelithiasis with occlusion of the cystic duct, jaundice cannot be thus explained unless we attribute to the substantive reflex contractions of the common bile duct and especially of the circular terminal muscle (*sphincter choledochi*) at the duodenal end the same rôle which we ascribe to the gall-bladder itself. Thereby the pressure of the biliary system exerts a degree of contraction sufficient for the propulsion of the bile into the circulation.

We see, therefore, that a satisfactory explanation of jaundice in all cases of gall-stone colic has not yet been given, and that in its production several factors are probably coöperative.

A symptom which is intimately connected with jaundice is *pruritus cutaneus,* which sometimes precedes the attack or follows it. With an existing jaundice, an increase of pruritus may announce an approaching attack. This symptom, markedly disagreeable, by causing scratching of the skin, may be the immediate forerunner of serious complications, such as furunculosis and secondary staphylococcus infection of the biliary passages, a condition which I saw in a patient from Salonica.

Local cutaneous jaundice in the form of a *greenish-black discoloration of the skin* in the right hypochondrium was observed some time prior to rupture of an abscess which had been formed by a perforation of a gall-bladder containing stones.

Xanthoma are not rare in jaundice; nevertheless I have frequently seen xanthoma in the *canthus of the eye or the lids* of gall-stone patients who had no jaundice, a finding to which I desire to attach a certain importance, particularly in the diagnosis of irregular cholelithiasis.

Symptoms on the part of the nervous system play an important rôle in cholelithiasis, and may appear during the paroxysm as well as in the separate phases of the disease, and may become prominent. It is self-evident that the nervous symptoms may vary according to whether we are dealing with a neurasthenic or hysterical individual, with a child or with an aged person.

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Marked psychical disturbances, attacks of mania, general convulsions, confusions and hallucinations, clonic spasms which begin in the muscles of the right side of the abdomen and extend to those of the extremity or affect the muscles at the back of the neck, and symptoms of hemiplegia may occur during the attack and so completely dominate the picture that, in exceptional cases, they may be the cause of death.

The *position of the patient* during the paroxysm may vary; some lie upon the back, others upon the side or the abdomen, others are bent over or kneel, propping the head upon the floor, and others again resort to a cradle motion resembling a mother nursing her child.

The patient that I saw showed during the attack the characteristic "arc de cercle" with hyperesthesia of the skin in the hepatic region and hysterical contractions.

Reflex paralyses, such as occur after affections of the bladder and uterus, were noted by Trousseau as sequels of gall-stones.

Headache is frequently mentioned in the literature of gall-stone disease. Frerichs observed cases in which marked headache, cardiac palpitation, active pulsation of the abdominal aorta, and epistaxis became prominent accompaniments of the attacks. Kehr saw cases in which migraine disappeared after the removal of gall-stones. Kraus found migraine a feature in the history of many gall-stone patients, and remarks that in some of these, on the first appearance of gall-stone colic the attacks of migraine became rarer or ceased entirely. I, too, have seen cases of the latter kind, but I believe that in most of them the finding of the two frequent pathologic conditions is a mere coincidence.

Headache in chronic jaundice is sometimes a phenomenon of cholemia; in other cases it is due to anatomical disease of the meninges, as, for example, pachymeningitis hemorrhagica or meningitis, which latter affection is not infrequently combined with infectious cholangitis, and forms the terminal phase of gall-stone disease. Therefore, in the course of a cholelithiasis, we may meet with severe cerebral symptoms, with delirium, with sopor and stupor. On this account, cholelithiasis is intimately related in a differentio-diagnostic respect to acute atrophy of the liver, in which the early appearance of psychical disturbances out of proportion to the other symptoms forms a striking diagnostic factor. Years ago, when an assistant in Bamberger's Clinic, I was called to Braun's Obstetrical Clinic to see a pregnant woman who was suddenly attacked by severe pain in the hepatic region accompanied by jaundice, so that at first she was thought to be suffering from gall-stones. Soon afterward delirium set in and the liver dulness progressively decreased. The autopsy showed acute yellow atrophy of the liver. Decrease of liver dulness in pregnancy is by no means easy to determine. Delirium and psychical disturbance may be observed in both diseases, and, besides, a chemical analysis of the urine for leucin and tyrosin cannot be immediately made at the bedside, nor is such test absolutely decisive, for cases of cholelithiasis have been observed in which leucin and tyrosin were present in the urine and were apparently due to focal necrosis of the liver cells.

Among the nervous symptoms, the *pains* which accompany the paroxysm of gall-stone colic form the most prominent symptom in the clinical picture. They begin usually 3 to 4 hours after a meal, therefore when the chief bulk of the acid food mass is passing into the duodenum, in contrast to the cardialgias of gastric ulcer and acute catarrh which appear soon after the intake of food. Owing to the active peristalsis of the biliary passages, the stone is caught in the neck of the gall-bladder, and is thus forced further into the cystic duct. When the spasm of the musculature of the gall-bladder ceases, the stone may regurgitate into the bladder, but, as a rule, it is carried onward until it finally reaches the duodenal mouth of the choledochus, and, eventually, the intestine. This transit of the stone from the gall-bladder to the duodenum in typical cholelithiasis usually runs its course under the well-known picture of gall-stone colic. The pains are here due to an acute dilatation of the biliary passages and irritation of their sensory nerve fibers, either by the stone or by the inspissation of bile caused by the stone, or also to an erosion of the mucous membrane by a stone with sharp edges.

Exacerbations of pain in an acute irritation of the biliary passages are caused by contractions of the musculature of the gall-bladder and of the biliary passages, analogous to uterine contractions in the period of expulsion. The greatest obstruction to the passage of stones is formed by the narrow cystic duct and the portio intestinalis of the choledochus.

The *two phases of pain* which theoretically are said to occur in the passage of the cystic duct and of the duodenal papilla in typical cholelithiasis, and which are not to be undervalued as diagnostic factors as two entirely distinct exacerbations of pain in the clinical picture of cholelithiasis as it has been described by some authors, may occur, but I have not as yet observed them in my cases. The cessation and the exacerbations of pain are due to contractions, and I believe that, analogous to rapidly succeeding uterine contractions, it is subsequently impossible to determine whether the first, the middle, or the last of the paroxysm was the most painful. I also believe that, in cholelithiasis, the line of demarcation between these two periods of pain, which is said to mark the passage of the stone from a narrow into a wider canal, is more theoretic than real.

According to Riedel, the paroxysms of pain in cholelithiasis, also the less marked pain at the onset of the attack, are not to be attributed to the impaction of stone in the cystic duct but to *cholecystitis.* The *vis a tergo,* which forces the stone into the cystic duct, is the pressure of the rapidly oncoming inflammatory exudate in the gall-bladder (*inflammatory dropsy of the gall-bladder*).

Riedel even attributes the colics which recur after the impaction of the stone to renewed inflammation of the gall-bladder.

The attacks of colic which accompany lithogenous closure of the cystic duct are designated by Riedel as *ineffective* if the stone does not pass the cystic duct, and are in contrast to *effectual* colic in which the stone, after having passed the cystic duct, enters the common duct, and finally reaches the duodenum.

Similarly, Riedel considers the attacks of colic including the jaundice which appears after the stone has reached the supraduodenal portions of the choledochus without occluding them and without producing stasis of bile, to be caused by a sudden inflammatory reaction of the mucous membrane to the foreign body.

We see, therefore, that even the mechanism of gall-stone colic is susceptible of different explanations.

In cases of cholelithiasis in which the colic manifests itself only by gastric spasm, vomiting and sensitiveness to pressure in the region of the gall-bladder, the exacerbations of pain upon movement in opposition to pain of other origin may have diagnostic importance.

A patient in the Clinic with cholelithiasis complained of exacerbations of pain when riding in poorly paved streets, and when jolting during the drive to the hospital, this being analogous to the onset of the pain in patients with stone in the bladder; another patient, who besides sensitiveness of the gall-bladder upon pressure also had friction sounds in the same region, remarked that upon using the right foot in jumping the pain in the gall-bladder region and above the right Poupart's ligament was particularly severe. Upon jumping on the left foot painful sensations were felt only in the gall-bladder region, and this was the case in rapid running or when lying upon the right side. In ascending a staircase the pains were more severe than in descending, but they were especially severe in jumping upon the right foot. The vertebral column, from the second to the sixth thoracic vertebra inclusive, was painful upon pressure, and within this zone of pain cutaneous hyperesthesia was present and more marked upon the right than upon the left side.

The *radiation of the pain* in cholelithiasis demands special notice. In classical cases, the pains begin in the region of the gall-bladder and radiate to the right shoulder. At the onset of the attack, some patients experience a peculiar chilliness and a drawing sensation along the back. Combined with this the region of the gall-bladder is sensitive to pressure. In contrast, there is quite an array of cases in which the localization of pain is an irregular one, and the pain radiates toward various regions of the body. The pains may be localized immediately below the xiphoid process without radiating toward the hypochondriac region, and without increasing upon pressure. Confusion with pretuberculous gastralgias, dyspepsia, ulcer and cancer of the stomach, and gastric crises is therefore possible.

Sometimes the pains are localized in the breast, in the thorax, in the esophagus, sometimes in the region of the mamma, so that the patients believe they are suffering from cancer of the breast. When the pain radiates to the abdomen, particularly around the navel, intestinal colic and enteralgia are simulated. The diagnosis between saturnine colic and gall-stone colic may, under some circumstances, cause difficulty, as in a case which came under my notice, occurring in a worker in artificial flowers; the patient simultaneously showed a lead line and mild jaundice. In women, affections of the genitalia, metritis, parametritis and the like are suggested to us, all the more so as gall-stone colic sometimes appears during menstruation. Frequently appendicitis comes into consideration, particularly if the vermiform process is bent upward or is even adherent to the gall-bladder.

The pains are stabbing or tearing; sometimes there is a girdle sensation. They may also wander with a change of position. The colicky pains in the right hypochondrium may be quite transitory or may last for several days. Radiation to the back or to the lumbar region, as occurs in carcinomata of the pancreas, in ulcer of the posterior wall of the stomach, in carcinoma of the stomach with metastases of the retroperitoneal glands, and in ulcer and carcinoma of the duodenum, is also observed in rare cases of cholelithiasis. In this affection the sensitiveness of the lumbar and dorsal vertebræ to pressure and to percussion is similar to that observed in ulcer of the stomach. But pain upon tapping the vertebra with the fingers appears to be more readily induced in gall-stone disease. In a patient, aged 68, added to the typical hepatic colic were burning pains radiating into the lumbar region and the region of the urinary bladder, with strangury and the discharge of dark urine depositing a profuse, red sediment. The patient suffered formerly from rheumatism of the joints of the hand and of the ankle-joints.

[Tenderness on palpation in the region of the gall-bladder, due to cholelithiasis or to cholecystitis, is characterized by a radiation of pain to the epigastrium. This fact I have observed in my own case and in many patients. Tenderness upon palpation in the right hypochondrium due to other causes may radiate or may not, but is not felt in the epigastrium.—Ed.]

In such cases, particularly if jaundice is absent, gall-stone colic is apt to be confounded with renal colic.

Irradiation of pain to the breast may be confounded with pleurodynia, rheumatism and intercostal neuralgia.

In the differential diagnosis of gall-stone colic and *nervous hepatic colic*, Th. Fuchs remarks tersely that nervous hepatic colic is invariably aggravated by a Carlsbad treatment. I have not as yet seen such an instance. The majority of cases which, upon superficial investigation, were supposed to be hepatic colic, proved later to be intercostal neuralgia. Some cases, too, of hysterical pseudo-appendicitis were found upon minute investigation to be nothing more than neuralgia of the 12th intercostal nerve and its ramus perforans anterior. In such cases the painfulness of the vertebral and lateral pressure points, as well as the presence of other nervous symptoms, and the hysterical stigmata are decisive.

In those cases in which the pain radiates toward the cardiac region and toward the left arm, the diagnosis may become very perplexing and even impossible. I observed a case of this radiation of the pain in a patient about 50 years of age, who, for some years, suffered from slight paroxysmal glycosuria, uraturia and oxaluria. Suddenly the pain appeared in the gall-bladder and thence radiated, the amount of sugar in the urine was increased to 4.5 per cent., the pulse rate declined to 45. Cardiac examination was negative except for a moderate accentuation of the second aortic sound. The diagnosis of competent physicians varied between angina pectoris, as a consequence of sclerosis of the coronary arteries, and cholelithiasis. The patient consulted me several months after the attack, and I made a diagnosis of gall-stones, chiefly for the reason that the gall-bladder was sensitive upon pressure, that the patient showed xanthomata bilaterally upon the eyelids, and because the glycosuria which had been observed after the attack but had now improved so that only traces of sugar were present could be best reconciled with the diagnosis of cholelithiasis. A marked increase in the excretion of sugar in angina pectoris of the diabetic is unknown to me.

In contrast to this I remember a case in Bamberger's Clinic, occurring in a woman, aged 35, who suffered from aortic insufficiency with coronary angina (angina pectoris). After such an attack, which ran its course with vomiting and slight jaundice, sensitiveness upon pressure over the liver appeared. The diagnosis was insufficiency of the aortic valve, sclerosis of the coronary arteries, cholelithiasis. The patient died soon afterward during an attack; the autopsy showed insufficiency of the aorta, narrowing of the coronary arteries, but *no* gall-stones.

These two cases prove that we may meet with the serious dilemma of being doubtful *whether angina pectoris or cholelithiasis* is present, and in this case every objective sign which may be of importance in the differential diagnosis between these affections must be minutely investigated.

Colic-like intestinal pains in the hypochondrium and right hypogastrium, with meteorism and constipation, may occur in cholelithiasis and be due to adhesion of the gall-bladder to the transverse and ascending colon. The development of adhesions of this kind is especially favored by a pendulous belly and hepatoptosis. Tearing and drawing pains in the hepatic region before each movement of the bowel, and pain radiating to the right shoulder, were observed by Kelling in a case of cholelithiasis in which the transverse colon formed adhesions with the border of the liver. After breaking up the adhesions, the pains disappeared.

Typical attacks of colic with chronic jaundice may be due to *simple adhesions without gall-stones.* In such a case, reported by Leichtenstern, the entire gall-bladder was atrophic, and firmly adherent to the surrounding tissues; no stones were present, neither in the cystic nor in the common duct. The adhesions were broken up, the jaundice disappeared soon after the operation, and the patient was completely cured.

Unlike many forms of pain arising in saturnine colic, the pain is usually unrelieved by pressure. Sometimes there is amelioration after the eructation of gas or after the passage of flatus and feces. Such cases may be confounded with hepatic colic and flatulent colic.

In the majority of cases the ingestion of food and fluid does not specially influence the pain. Though it sometimes happens in gall-stones that the attack of pain develops immediately after the intake of food, and erroneously leads to the diagnosis of ulcer of the stomach, and although, on the other hand, in the case of an ulcer with decided secretion of gastric juice and hyperchloria, not rarely the pain ceases after the ingestion of food, whereby the HCl is combined, yet the influence of the character of the food and the fluid ingested must not be undervalued. Patients who formerly experienced no difficulty in taking food which was highly spiced and seasoned now find themselves unable to partake of this without severe gastric pain; this circumstance, in a given case in which the diagnosis hinges between ulcer and cholelithiasis, decides us in favor of ulcer, as in a case I observed, which, by the later occurrence of hematemesis, proved to be ulcer. The pain is sometimes augmented after drinking sour wine, and this may, under some circumstances, favor ulcer, while a patient with gall-stone, who was known to me, after taking quite a quantity of wine, noted amelioration of her pains.

Gall-stone patients can sometimes digest heavy and irritating foods very well, but there may be idiosyncrasies for certain foods. A patient whom I knew, according to his account, invariably had an attack of gall-stone colic after eating fish with mayonnaise; some drugs, pepsin and hydrochloric acid, administered for gastric difficulty of doubtful origin, may be a guide in the diagnosis of these cases which are so frequent in practice. While I have seen no noteworthy influence upon the symptoms of cholelithiasis after the administration of pepsin and hydrochloric acid, I saw a case in which this remedy was given for cardialgia; the pains increased and a few days after the use of the remedy hematemesis occurred. This observation led us to be more cautious in the use of an active pepsin in doubtful cases; and it may be noted that even such a therapeutic experiment, which was here accidental, may be of value in those which are obscure.

The nocturnal exacerbations of pain which occur in a great number of cases of cholelithiasis are explained by Kehr as being due to the accumulation of bile in the gall-bladder. In biliary fistula we observe that the excretion of bile during the day is very slight, but it is profuse during the night. "During the night when a person sleeps and no food is taken, bile collects in the gall-bladder." Although this remark and explanation of Kehr are quite correct, and may be utilized in the differential diagnosis between gastric ulcer and cholelithiasis, inasmuch as pain from ulcer is rarely present in an empty stomach or at night, while gall-stone colic frequently occurs at night with an empty stomach, no absolute proof can be attached to this symptom, the less so as pains, particularly at night (Hesse) have been observed in ulcer, as well as amelioration of the pain on the ingestion of food, especially when there is simultaneously existing hypersecretion. The nocturnal appearance of pain is emphasized in the rare syphilitic form of gastric ulcer.

I have not tried the effect of a *hunger cure* carried out systematically for several days in suitable cases for diagnostic purposes, although it is reasonable to assume that such an attempt is justified in complicated cases, especially when the question of operation is under consideration. *A priori,* it must be taken for granted that, for example, the gastric crises of the tabetic are uninfluenced by such a hunger cure; the same is very likely true to some extent of pains due to stone in the biliary passages, while in ulcer, because of insufficient gastric secretion in the overwhelming majority of cases, the pain either ameliorates considerably or ceases entirely.

The variable *intensity of the pains* in cholelithiasis is not always in proportion to the other reflex symptoms on the part of the stomach, for example, vomiting. In a patient observed in the Clinic, a day laborer, John Sch., aged 57, the paroxysms of pain in the right, upper abdominal region, running their course with jaundice, were of such extraordinary severity that the patient writhed in bed, moaning. In this severe paroxysm of pain which, according to the accounts of the patient, was much more severe than the earlier ones in which vomiting occurred, symptoms on the part of the stomach were entirely absent.

The pains which are due to pericholecystitis, perihepatitis, circumscribed abscesses or rupture into the peritoneum, belong to the realm of *peritonitis.*

It must not be forgotten that abdominal pain and pain in the righ hypochondrium during menstruation may also be due to gall-stone colic The same is also true in pregnancy.

Cases have been reported in which, at the normal termination of pregnancy, gall-stone or renal colics have been mistaken for labor pains; al in childbed, when pains arise in the abdomen with vomiting, fever, an chills, cholelithiasis may be simulated. Here only an exact history, t appearance of pain a few hours after meals, the pulse, and a most thoroug examination of the gall-bladder region and the right hypochondrium w lead to the diagnosis of a complicating cholelithiasis.

The *differential diagnosis between gall-stones and pancreatic stones*, especially as jaundice may occur also in the latter affection, causes great perplexity.

Simultaneous diabetes, or that occurring in combination with colic, fatty stools, azoturia, and, finally, the passage of stones of calcium carbonate and phosphate, are important diagnostic points of support.

When *tabetic gastric crises and jaundice occur simultaneously*, the diagnosis is sometimes only possible by the careful discrimination of the individual symptoms.

I saw a patient who for four years suffered from attacks of pain which occurred irregularly at intervals of from three weeks to three months; the patella tendon reflex was absent; there was immobility of the pupil. During an attack a year ago jaundice lasted for two weeks. A person suffering from tabes may, therefore, simultaneously have cholelithiasis, or, by a temporary indigestion, catarrhal jaundice may be superadded.

Prior to this we have considered two of the most frequent and marked symptoms of cholelithiasis, *jaundice and pain*. Just as there are cases of cholelithiasis without jaundice, there are others, with or without jaundice, which run their course with freedom from pain. Cholelithiasis without pain is certainly a very rare form. Complete but painless occlusion of the choledochus by stone may appear under the guise of catarrhal jaundice or of a neoplasm at the porta hepatis. In chronic empyema of the gall-bladder, local pain may be very slight or entirely absent.

In these *indolent* forms of cholelithiasis, another element becomes prominent and almost substitutes for the attacks of colic, namely, *fever*, which, therefore, may substantively be of great diagnostic significance.

In the *aged*, in whom attacks of gall-stone colic need not necessarily cause serious disturbance, a mere chill may be the only manifestation of the disease.

In other cases, chills may alternate with pain in the right hypochondrium. The chill may run its course with collapse, cyanosis of the lips and cold extremities, followed by fever with or without sweating. The paroxysms of fever in cholelithiasis begin, as a rule, a few hours after eating, therefore in the evening or during the night, in contrast with those of malarial attacks.

Even when the stones are large and completely obstruct the common duct, this intermittent fever may dominate the clinical picture. As an assistant in Bamberger's Clinic, I once saw a patient with obstructive jaundice and intermittent fever, who, according to her report, had *never* suffered from colic, and who, upon *objective* examination, showed no local sensitiveness in the hepatic region; at the autopsy, a stone the size of a walnut obstructing the common duct was found, while no signs of a preceding lithiasis were present in the gall-bladder. Bamberger regarded this case as a secondary gall-stone formation in the common duct, developed by the apposition of the cholesterin to a stone which had wandered into the common duct without giving rise to symptoms, and the painlessness of its course as due to the mucous membrane becoming accustomed to the foreign body slowly growing *in situ.*

The *onset of the pain with chills* is important in the diagnosis between gall-stone colic and cardialgia in ulcer of the stomach. Rises in temperature in ulcer of the stomach may be caused by circumscribed peritonitis. The preceding gastric disturbances, the dependence of pain upon a co-existing peritonitis, the absence of chills will also aid in the diagnosis of such complicated cases.

Doubt may arise in the differentiation of *duodenal ulcers* which develop upon the basis of a *paroxysmal hemoglobinuria.* An interesting case of this kind was admitted to the Clinic in May, 1895, with a diagnosis of ulcer of the stomach. The patient was always well until July, 1892 when he was taken ill after having been for two days previously exceedingly pale, and, according to the statement of his wife, the sclera havin become yellow. During a journey he was attacked by nausea, sever gastric pains, vomiting of greenish masses, and collapse without loss o consciousness. The vomiting was repeated once, but did not recur. O the other hand there was gastric spasm, loss of appetite, acid eructatio and a feeling of heat; the bowels were constipated. Melena and hematen esis never occurred; the urine was blood red in color. A few days aft the patient had left his bed, he completely recovered.

Up to January, 1895, the patient had three such attacks. In the la which caused him to come to the Hospital, there was pallor and loss appetite, the gastric pains recurred and radiated toward the lumbar regi to the right of the navel. He vomited greenish masses of an acid odo there was pyrosis and eructation. Acid food increased the gastric d tress, while the pains were lessened by sodium bicarbonate. The objecti examination showed high-grade anemia and a moderate degree of gast dilatation. The majority of the symptoms, therefore, indicated ulcer the stomach, and two only were calculated to obscure the diagnosis: On one hand, the chill which actually introduced the first attack, and, the other hand, his wife's report of the yellow discoloration of the scl in the first attack. These two symptoms favored cholelithiasis. Nev theless, our tentative diagnosis was: Ulcer of the stomach.

The patient left the Hospital. Four months later he returned to Clinic with the same symptoms, and, in addition, a nephritis and he globinuria. The attack was introduced by a chill. Several months l the patient died. The autopsy showed *ulcer of the duodenum.* We therefore, that with the symptom-complex obtained by the history—dialgia, chills and jaundice instead of the looked-for gall-stones, an expected combination of two rare affections prevented a correct diagn In the majority of cases, however, the occurrence of rare cases of kind is not calculated to lessen the clinical value of the previously mentioned triad of symptoms in the diagnosis of cholelithiasis.

The chills which accompany fever and gall-stone colic were formerly believed to be reflex and due to the influence of the heat-regulating center. On this conception of fever as a pain reflex, Bamberger chiefly based his opinion of the favorable action of morphin, which, in some cases, by relieving the pain, simultaneously had an antipyretic effect. In many other cases, however, we are compelled to assume a pre-existing, or subsequent, infection of the biliary passages. This is true, in particular, of those cases in which the febrile paroxysm is not combined with an attack of pain, and even more so in those instances in which the febrile attack is the only manifestation of cholelithiasis. Infection of the biliary passages may occur in various ways, one of which is that the bacteria find a passage directly from the intestine into the common duct. If, by disease of the biliary passages, a *locus minoris resistentiæ* is formed for the entrance of bacteria, the latter, on the other hand, is favored by dilatation of the biliary passages and the opening of the duodenal mouth, and thus infection with intestinal bacteria takes place. There is also the possibility of infection with virulent bacteria from the oral and pharyngeal cavities, for example, from tonsillar abscesses the discharges of which are swallowed with the saliva. Infection of the biliary passages may also occur via the circulation from distant foci in the organism, as from otitis media and angina tonsillaris, etc., as is also assumed to be the case in appendicitis. These manifold sources of infection also explain the bacterial polymorphic nature of infectious cholecystitis and cholangitis (bacterium coli, diplococci, streptococci, staphylococci and typhoid bacilli).

The attacks of fever which accompany paroxysms of pain in cholelithiasis are, at present, almost universally referred to infection of the biliary passages, and are not considered as reflex fever, as was formerly the case. Although we must admit the possibility that a typical colic fever in which chills, fever and paroxysms of pain go hand in hand may be a nervous reflex fever, analogous to the febrile attacks in paroxysmal hemoglobinuria, or a purely toxic fever due to pyrogenous derivatives, perhaps of hemoglobin, nevertheless, it cannot be denied that, in the overwhelming majority of cases of *intermittent hepatic fever* infectious processes in the biliary passages and in the liver are the causative factors. This is particularly true of the cases in which fever appears in a substantive form and independent of the colicky pain, and further, of those cases in which there are objective signs of a pericholecystitis or perihepatitis, and, finally, when we have, as accompanying symptoms of the fever, signs of a local inflammation or infection in other organs removed from the liver, or in the blood, as, for example, enlargement of the spleen, nephritis, endocarditis, pericarditis, bacteriemia and septic leukocytosis.

There are many transitional stages between the periodic intermittent *gall bladder*, yet no stones are found at the operation. Gersuny performed an operation on the wife of an apothecary who had irregularly recurring colic, chills with fever, enlargement of the liver, pain in the region of the gall bladder, splenic tumor and gastric dilatation. He found the gall-bladder contracted, no cholangitis, no stone; but the gall-bladder was adherent to the duodenum, and there was gastric dilatation. After breaking up the adhesions the fever disappeared and recovery took place. The process may, perhaps, have been due to retention of bile in the adherent gall bladder, which formed a favorable culture media for the microbes there present. By the succeeding restoration of free communication between the gall bladder and the cystic duct, the condition was relieved.

As a cause of the development of intermittent fever, chronic morphinism is yet to be discussed. A case of this kind is known to me in a morphin habitue in whom the most minute investigation of the various organs revealed no cause for the fever. Examination of the urine showed acetone and a conspicuous increase of uric acid excretion to 40 grams per day. In gall-stone patients who, in consequence of preceding colic, have become morphin habitués, this possibility must be borne in mind, and an examination be made of the urine, which, in hepatic fever, shows a decrease in the excretion of nitrogen.

The foregoing remarks prove conclusively the many-sided nature of [illegible] and how easily errors in diagnosis may be made in cases [illegible] without pain or jaundice.

A [illegible] of the biliary passages are not accompanied by fever [illegible] with threatening [illegible] and pain, particu[illegible] observed par[illegible]

[illegible] disease, eithe[illegible]

[illegible] cases met with [illegible] regard to the [illegible] produced by the operation shows no cause for the condition, necessitates the utmost caution on the part of the surgeon.

A dancer, aged 22, was admitted to the Hospital—special stress must be laid upon her occupation, since, in the etiology of cholelithiasis, a sedentary habit is considered to be predisposing—after an undoubted cholelithiasis with alternating jaundice which had existed for seven weeks; there were cutaneous hemorrhages upon the hands and feet, and after scratching off a wart ad nates, a hemorrhage occurred which could scarcely be controlled.

In an examination of the neck, the *jugular glands* must not be overlooked since they are of importance in the differential diagnosis of carcinomata of the biliary passages and of the liver. It is sometimes advantageous to have the patient cough during the examination. Deeply situated, isolated glands behind the clavicle become prominent during coughing, and are thus made susceptible to palpation.

Examination of the lungs is important inasmuch as, according to some authors, tuberculosis predisposes to the formation of gall-stones. In my experience, however, clinically well-developed cholelithiasis, as well as cholangitis, is an extremely rare complication of tuberculosis, and this is all the more remarkable as, in ulcerative phthisis, bacteriemia (streptococcia and staphylococcia), which is so very frequent, furnishes a fertile soil for infection of the biliary passages. I have found, on the contrary, that the tubercular have a certain immunity from gall-stone formation, and patients with cholelithiasis from tuberculosis.

Attacks of cough occurring in paroxysms (the *tussis hepatica* of the older authors) followed by aphonia, hoarseness and severe pains in the back of the neck, have been observed in cholelithiasis and have disappeared after operative removal of the stones.

Dyspnea, sensations of fear, even attacks of suffocation, also occur as symptoms of gall-stone colic, and may be due to reflex spasm of the muscles of respiration.

Immotility of diaphragmatic respiration, therefore, the costal respiration type, in adults, particularly in men, is an important symptom in peritonitis, in contrast to the respiration in gall-stone colic; while small children who, as is well known, normally almost exclusively show the abdominal type of respiration, present the costal type of respiration, and this symptom is an important sign in the differential diagnosis of infantile gall-stone colic, in opposition to intestinal colic of other origin. Sensitiveness to pressure over the gall-bladder, especially when in a warm bath, and muscular rigidity on the right side of the abdomen, combined with the preceding symptoms, form additional proof. Cholelithiasis under twelve years of age, and even under fifteen years, belongs, in fact, among the greatest rarities.

In children, provided jaundice does not point to the possibility of gall-stones, epigastric pain, vomiting and convulsions are many-sided symptoms, and this doubtless will sufficiently explain the fact that the disease is usually overlooked in early childhood.

In the aged, as the result of gall-stone colic, which, as has been stated, sometimes manifests itself only by chilliness and fever, a *congestion of the lower lobe of the right lung* is observed, which, when combined with cough, muco-viscid expectoration and numerous crepitant and fine moist râles at the base of the right lung, also with fever and pain, may be mistaken for pneumonia. This hyperemia of the lungs, appearing under the guise of pneumonia, may clear up in two or three days after the rudimentary attack of colic. It may, however, also form the starting point of a secondary pneumonia. In cases in which cholelithiasis, by infection of the biliary passages, has led to suppurative cholangitis, biliary abscesses of the liver, or subphrenic suppuration, important diagnostic processes may take place at the base of the right lung: unilateral, high position of the diaphragm with retraction of the lung, pleurisy, symptoms of putrefaction of the respired air and, a few days prior to rupture, distressing cough with purulent, bile-stained expectoration and râles at the base of the right lung. Nevertheless, rupture of the diaphragm and pleura, as well as subphrenic abscess, with the formation of a pyopneumothorax, supraphrenicus and subphrenicus, are very rare episodes in the course of infectious cholelithiasis, in contrast to echinococcus of the liver, which, as is well known, not infrequently forces a passage into the lungs by perforation of the biliary channels and of the diaphragm.

In the course of cholelithiasis symptoms [illegible] of *the heart* and circulation [illegible] the situation to such an extent that, in some cases, [illegible] affected by cardiac disease. Collapse [illegible] during the paroxysms of pain [illegible] particularly in women, and [illegible] as a vaso-motor reflex, or in [illegible] Cardiac arrhythmia, dyspnea, [illegible] sound, dilatation of the right heart [illegible] relative tricuspid [illegible] with cyanosis, and a [illegible] of an attack of [illegible] sense; with [illegible] transmitted by irritation [illegible] pneumogastric [illegible]

The pulse [illegible] normal or [illegible] cases; in [illegible] simultaneous inflammation of the gall-bladder and [illegible] frequent pulse, naturally, may also occur in general peritonitis and in gastric and intestinal hemorrhages, as well as in septic and pyemic fever in gall-stone patients, and during the apyrexia, especially if an infection from the biliary passages and the liver localizes itself in the heart as a pericarditis, endocarditis or myocarditis.

The tension and configuration of the *abdomen* may furnish objective signs of importance in the diagnosis, particularly as regards the complications of cholelithiasis. *Contraction of the abdominal muscles,* mainly upon the right side, which the French designate as défense musculaire, frequently accompanies the attack of colic; but there are also cases in which the abdomen, in spite of severe pain, is everywhere soft, sometimes even showing meteorism. *Meteorism* which occurs in jaundice in consequence of intestinal acholia, or with a co-existing peritonitis in an obstruction of the intestine by stones, or in intestinal stenosis in consequence of adhesions, is easy to explain. *Ascites* may occur in cholelithiasis by occlusion of the portal vein. The irritation of the incarcerated stone in the common duct produces consecutive induration in the hepato-duodenal ligament, and, as a further result, thrombosis of the portal vein and an ulcerative perforation of the walls by which stones enter its lumen.

In this group belongs the case that is everywhere quoted of Ignatius de Loyola, in whose portal vein Realdus Columbus found three gall-stones. In general, however, ascites in cholelithiasis is rare in comparison with carcinoma at the porta hepatis, which, by frequent compression of the portal vein, as well as by secondary carcinosis of the peritoneum, leads to ascites. This is particularly true of the cases running their course with obstructive jaundice, just as an ascites developing simultaneously with jaundice almost always indicates an abdominal neoplasm.

In gall-stone patients the *panniculus adiposis* (subcutaneous fat) is often markedly developed, particularly in the abdomen, which naturally renders the physical examination difficult.

Peter observed, during the attacks of colic, a *local rise of temperature in the right hypochondrium.* The temperature in this region may be as high as that of the axilla, or exceed it, while, under normal circumstances, it is two to five degrees lower than that of the axillary temperature. In the gastric crises of tabes such a local hyperthermia in the right hypochondrium is not observed.

In cholelithiasis the *liver* is either of normal size or enlarged, and this is due either to hyperemia, biliary stasis, abscess formation, or secondary carcinomatosis. In cases in which cholelithiasis occurs in cardiac patients, according to my experience a very frequent combination, the diagnosis of acute enlargement of the liver may cause perplexity, and the disease may either be attributed to gall-stone affection or be considered as acute stasis hyperemia. The pain may be due to the gall-stone or to tension of the capsule of the liver. I remember such a case of cholelithiasis in a patient suffering from mitral insufficiency in which at first I wavered in the diagnosis and was in doubt as to the therapy to be instituted. Digitalis was without effect, but a carefully conducted treatment at Carlsbad caused the swelling and painfulness of the liver to disappear. The diagnosis of cholelithiasis was confirmed by the passage of small stones.

Enlargement of the liver, which is frequently observed in patients with gall-stones without jaundice, may be due to parenchymatous swelling or simultaneously active hyperemia of the organ, and this is, in fact, the view of French clinicians (*congestion hepatique*).

If to the cholecystitis calculosa an infectious cholangitis be added, the latter sufficiently explains the enlargement of the liver. There are, however, cases characterized by irregular fever, intermittent enlargement of the liver with sensitiveness upon pressure, yet without well-developed colic or jaundice. This rapid increase in the volume of the liver can hardly be explained except by the existence of acute hyperemia. This is illustrated by a case known to me in which, at the onset, a diagnosis was made of infiltrating carcinoma of the liver; but the rapid decrease in the size of the organ and the absence of pain scarcely permitted a different interpretation than that of cholelithiasis intrahepatica.

Diffused pain in the enlarged liver, the symptoms otherwise being pyemic, points to suppurative cholangitis, while a local sensitiveness tc pressure outside the gall-bladder region, particularly when the pain radiates to the right shoulder, is found in abscess.

An enlargement of the liver with an *induration hard as wood,* chiefl in the region of the incisura, points to a carcinoma of the gall-bladde which has proliferated into the liver; particularly when cirrhosis is excluded and enlargement of the spleen is absent.

Elongation of the suspensory ligament in consequence of extreme dil tation of the gall-bladder, especially when hepatoptosis already exists, ma produce a *wandering liver* showing extraordinary·power of motion. *Per hepatitis* in cholelithiasis demands especial attention. In a case observ in the Clinic, a shoemaker, aged 53, had intermittent, irregular fever wi chills, rises in temperature above 102.2° F. combined with colic-like pai in the liver, a conspicuously rapid pulse even in the afebrile and painle intervals, varying jaundice, with only occasionally bile-stained stools; d tributed perihepatitis, manifesting itself over the whole extent of the liv by friction sounds, was the dominant feature of the clinical picture. T autopsy showed obstruction of the choledochus due to stone, dilatation the upper portion of the same, of the hepatic duct, and of the bile du of the liver, ulceration of the wall of the common bile duct, and the forn tion of multiple abscesses from the size of a pea to the size of a be containing pus, mostly grayish yellow, but still bile-stained, and the wa of which consisted of discolored hepatic tissue. In regard to the ausc tatory findings during life, such a general perihepatitis, if accompan by other severe symptoms, must awaken suspicion that in the interior the liver a severe infection of the biliary passages has taken place with the formation of abscesses.

Compression of the hepatic artery may produce *aneurysm*. Such a case was observed by Schmidt. Here death occurred with the symptoms of fatal gastric and intestinal hemorrhage; the gall-bladder, filled with stones, communicated with the aneurysm.

The circumstance that aneurysms of the hepatic region sometimes produce murmurs reminds us again that *auscultation of this region* should not be neglected.

In examination of the liver the first point of attack is, naturally, the breeding place of gall-stones—*the gall-bladder*. If this organ, at the beginning of the attack, contracts about its fluid or solid contents, and provided the abdominal muscles are not too tense, it is found as a globular, sometimes fluctuating, tumor which may be palpated through the abdominal wall. Some patients themselves note the intermittent contractions of the gall-bladder, and in their history report this to be as though a tumor were being inflated in the right hypochondrium, but after cessation of the pain this disappears.

These sensations of the patients are very difficult to explain, as the intermittent swelling in the right hypochondrium may also be due to an accompanying acute enlargement of the liver. The contractions of the gall-bladder may be extraordinarily severe. In the case of a man who succumbed to reflex spasms during an attack, Gerhardt found impacted at the entrance of the cystic duct a large stone, splinters of which had broken off from the anterior end, and had penetrated the mucous membrane. The *gall-bladder tumor* moves upon inspiration, and is not fixed upon expiration, which is explained by its direct connection with the liver.

The *lateral, pendulum-like, displaceability of the tumor* is important in the differential diagnosis of tumors of the gall-bladder and of the kidney. In congenital elongation of the cystic duct the tumor may be very movable. Such a gall-bladder, displaced and filled with stones, and very painful, may be mistaken for wandering kidney.

In a case of Fournier's which occurred in a young woman, a diagnosis was made of myelitis, neuralgia, abscess of the liver and floating kidney, but, over the right kidney, an irregular yellowish tumor the size of an orange was found, which was formed by the gall-bladder filled with stones. The hypertrophic ductus choledochus and cystic duct had a length of from 15–25 cm., which explains the dislocation of the gall-bladder. But the tumor may also be immobile, and adhere to the convex surface of the liver, which, in certain cases, may lead to the assumption of hepatic echinococcus and abscess.

A tumor of the gall-bladder, which is distended in consequence of closure of the cystic duct, cannot, naturally, become smaller by manual compression. If, however, the obstruction in the cystic duct can be overcome by this manipulation, a decrease in the size of the tumor may be brought about. A few instances of a gall-bladder of this kind, which may be expressed by paralysis or atrophy of the musculature, have been observed. Great distention of the gall-bladder may, in rare cases, produce permanent jaundice by compression of the hepatic or common gall-ducts. In lithogenous or cicatricial closure of the cystic duct the behavior of the gall-bladder depends, in the main, upon the texture of its mucous membrane. If the mucous membrane remains intact, secretion continues, and *dropsy of the gall-bladder* develops. In the opposite case, with marked damage of the mucous membrane the gall-bladder is obliterated, and becomes adherent to its surroundings. Stones may thus become encapsulated in the gall-bladder and form a harmless *caput mortuum*, as, for instance, an encapsulated bullet after a gun-shot wound. A gall-bladder of this kind filled with stones may sometimes be felt as a hard body in the incisura hepatis. Therefore, a lithogenous closure of the cystic duct is an important condition, especially for the surgeon, who most frequently deals with such adherent gall-bladders, less so for the internal clinician, who looks upon obliteration of the gall-bladder and encapsulation of the stones as a favorable conclusion of irregular cholelithiasis.

In lithogenous occlusion of the common gall-duct dilatation of the gall-bladder is very rarely found; usually it follows a preceding cholecystitis, while in carcinomatous compression or obstruction of the choledochus (excepting lithogenous choledochus cancer) distention of the gall-bladder is the rule. To this rule, which was established by Courvoisier, there are, however, many exceptions in either direction if we bear in mind that the condition of the gall-bladder depends, in the main, upon the extent of the mucous membrane lesion.

Impermeability of the cystic duct may be due to a lymph-gland lying alongside of it, which, according to Riedel, may be palpated at the operation as a small tumor and may be confounded with stones or cicatrices.

Acute perforation from pressure necrosis of the wall and ulcerative perforations of the gall-bladder sometimes leads to the formation of abscesses in the surroundings of the same, producing cavities, which are limited by the peritoneal, perihepatic, and pericystitic tissue formation. These cavities communicate with the gall-bladder, and may also perforate the abdominal wall, discharging greenish purulent fluid, and frequently stones, in large quantities. With an elongated gall-bladder, abscesses may thus form in the right iliac fossa. Such a deeply-situated, purulent cholecystitis may readily be confounded with perityphlitis, and, inversely, a vermiform process curving upward, or even adherent to the gall-bladder in the gall-bladder region, may resemble perityphlitis.

The tumors of the gall-bladder which are filled with stones may sometimes, by adhesion and displacement of the omentum, appear to be hard, and of considerable size.

A hard tumor in the gall-bladder region may be formed by a tongue-like process of the liver, if a corset lobe follows the direction of the slowly enlarging gall-bladder, and reaches beyond it. This so-called *Riedel's lobe* may give rise to difficulties in the differential diagnosis between cholelithiasis and syphilis of the liver. Echinococci bordering upon the portions of the liver in the incisura hepatis (original observation) may also lead to perplexity.

An excessive hardness of the distinctly enlarged and usually nodular gall-bladder, which is generally sensitive to pressure, and which then continues to the liver (*infiltration ligneuse* of the French), points to carcinoma of the gall-bladder, particularly when the infiltration of the gall-bladder is continuous to the liver. Hard nodules in the latter confirm the diagnosis. The lancinating character of the pains, meteorism, periodic vomiting, progressive jaundice and cachexia, a repugnance to meat and fatty foods, and monotonous anorexia which at the same time becomes prominent, which is permanent and invariable, enable us to differentiate typical cases of carcinoma of the gall-bladder from the lithiasis of old persons in which the appetite may be capricious, but in which, in the majority of cases, the painful exacerbations of the affection leave no doubt of its dependence upon gastric disturbance. Yet, even here, there are deviations from the ordinary type, as in a case observed in my Clinic.

Johanna A., a woman aged 56, was taken ill with burning pain in the gastric region and jaundice, and was treated by her family physician for gall-stones. During the entire course of the disease the patient had a good appetite, although it was somewhat decreased. Free HCl was determinable in the vomitus, pepsin was positive. Fifty per cent. of albumin solution was digested. Digestive leukocytosis present. The steadily growing tumor of the gall-bladder with increasing jaundice was shown at the autopsy to be due to an *endotheliosarcoma* of the gall-bladder.

While secondary stone formation in primary lithiasis of the gall-bladder is very frequent, we find in primary cancer of the gall-bladder, provided necrotic particles form the basis for stone formation and contraction of the gall-bladder is inhibited, that it is somewhat rare—therefore, in spite of biliary stasis and the presence of centers of crystallization, no gall-stones; in the rare instances of primary carcinoma of the gall-bladder, they did not consist of cholesterin but of calcium phosphate and carbonate.

In cases that are difficult of solution, in which the diagnosis between a tumor of the gall-bladder and one of the transverse colon must be made, naturally, all of the manipulations which are employed in the differential diagnosis of abdominal tumors, for example, inflation of the stomach and intestines with air and with water, must be employed. Upon bimanual examination in the left lateral position, and by making strong pressure against the right hypochondrium with the right hand, we sometimes succeed in pressing the gall-bladder to the right wall, and thus make it possible for the left hand to palpate the organ.

If the abdominal walls are flaccid, the gall-bladder is sometimes visible, which is, perhaps, impossible in the recumbent posture.

By adhesion of the stone-containing gall-bladder to the large omentum, the transverse colon may be lacerated, constipation, meteorism and the like may be the further consequences, and an atypical cholelithiasis may thus present itself under the picture of intestinal or pyloric stenosis.

By a contracted and displaced gall-bladder, and by pericholecystitic adhesions, the stomach, the duodenum, the gall-bladder, and the biliary passages may be torn; according to the tension of these organs, vomiting may be produced, and, by the bending of the cystic duct, intermittent colic.

From these adhesions between the stomach and biliary passages which resist all internal treatment, the *gastric symptoms* become prominent, and among the digestive disturbances which play such an important rôle i the picture of cholelithiasis *vomiting* is of special diagnostic import. Vomiting with severe retching, as a rule, accompanies the attack of colic, an generally produces little, if any, amelioration of the pain; while, in acut dyspepsia, gastralgia and ulcer, the pain usually ceases after vomitin This vomiting in gall-stone affection usually occurs several hours aft eating, but not invariably. In stone obstruction of the duodenal end the common bile duct, irritation of the papilla of Vater may cause pai in the gastric region as well as vomiting soon after the ingestion of foo the condition simulating ulcer of the stomach or ulcer of the duodenum.

In addition, it must be emphasized that, according to Fleiner, in co sequence of cholecystitis calculosa, actual gastric and intestinal ulcers m develop, as well as those of the large intestine, beginning from the sero which may perforate. Therefore, an accurate history, particularly in gard to the localization and radiation of the pain, should be obtained. may be easily understood, however, and has already been observed, t true ulcer of the stomach, confirmed by operation, and particularly ul of the duodenum combined with jaundice and pains radiating toward liver, has been mistaken for cholelithiasis.

Vomiting in gall-stones may occur in the early morning hours, vomitus matutinus. A cook, aged 34, Rosalia Sch., who admitted excessive use of alcohol, had attacks of gall-stone colic due to a tu of the gall-bladder of the size of a walnut. Colic only occurred a midnight or toward the early morning hours, and ended with vomi which appeared soon after arising from bed while the stomach was empty. Frequently the origin of matutinal vomiting is very obscure. though in the history of women suffering from gall-stones the abus alcohol is sometimes expressly emphasized, yet it is, nevertheless, a spicuous fact that in classic atrophic alcoholic cirrhosis, according to observation, cholelithiasis neither shows itself as a complication during life, nor is it found at the autopsy to be auxiliary.

This experience all the more furnishes food for thought inasmuch as in cirrhosis, in spite of the profound damage of the liver-cells, the bile in the gall-bladder shows no conspicuous anomaly, at least, none in appearance.

Vomiting in gall-stone disease may be alimentary, bilious, stercobilious, or stercoral.

The gastric contents are usually normal or show diminished acidity; sometimes, however, there is even hyperchlorhydria, especially if the gall-bladder is adherent to the pylorus, secondary gastric dilatation, and irritation of the gastric mucous membrane by the retention of the ingesta.

Whether the gastric contents are vomited, or the bilious contents of the duodenum, depends mainly upon the intensity of the act of vomiting. As a rule, vomiting is so violent in gall-stone colic that the ejected material is bilious. In infrapapillary duodenal stenosis due to laceration and compression of the duodenum, the appearance of bile in the vomited material may be readily understood. On the other hand, in occlusion of the choledochus, the vomited material will contain no bile.

Uncontrollable vomiting with intense pain, great anxiety, constipation and meteorism, particularly in the hepatic region, is usual in perforation of the gall-bladder and in biliary abscesses. Uncontrollable vomiting of ingested food after the intake of nourishment, with severe pain in the hypochondrium radiating to the right shoulder and back, stubborn constipation, unquenchable thirst, and singultus were the clinical symptoms in a case of duodenal stenosis observed by Fauconneau-Dufresne which was due to compression of the tumor by the gall-bladder.

Stercobilious vomiting is almost always a sign of disturbance in the lumen of the intestine, and the appearance of stercobilin in the vomitus is of serious import in the course of cholelithiasis. I emphasize this because *intestinal occlusion* due to incarceration of stones in the intestine may, on the one hand, appear under the picture of *ileus* and run its course as the first manifestation of a latent cholelithiasis, or, on the other hand, gall-stones may appear under the mask of an acute intestinal stenosis, in which laparotomy reveals no obstruction of the intestine, neither invagination, torsion, nor stones, no peritonitis and no perforation; at the autopsy a large gall-stone is found in the common duct. These cases resemble those of nephrolithiasis, running their course under the picture of intestinal occlusion, of which I have seen 3 examples, and in this category also belongs the case of a fat woman, aged 58, who suffered from severe pain in the upper abdominal region, chills, fever and bilious vomiting. An irreducible umbilical hernia was found, painful upon pressure, which had, according to report, existed for some years. A diagnosis was accordingly made of incarcerated umbilical hernia. Laparotomy revealed cholecystitis nausea and nothing abnormal in the umbilical hernia. Removal of the stone was followed by recovery.

Large stones, which by perforation of the common duct or the gall-bladder reach the intestine, may produce symptoms of occlusion, and either soon after their entrance or a long time afterward, even weeks, months or years, severe symptoms of intestinal stenosis may appear. After prolonged retention in the intestine owing to peristaltic and antiperistaltic movements, they may become fixed in some portion by spastic contraction, either in the duodenum, jejunum or ileum, usually in the region of the ileo-cecal valve. Large stones may also reach the cecum and the large intestine after dilatation or ulceration of Bauhin's valve (ileo-cecal). In this manner even stones the size of a hen's egg may be discharged with the feces. In other cases, they lead to ulceration or perforation of the intestine with all their consequences. Rupture may occur directly into the peritoneal cavity and into cavities formed by preceding inflammation, and thus circumscribed abscesses develop. This shows how valuable for correct diagnosis are many obscure cases of intestinal occlusion or ulceration in the ileo-cecal region, and how important is an accurate history which may eventually point to a preceding cholelithiasis. This is all the more true since the stones which have reached the intestine by perforation may have been present for months and even for years in the intestinal mucous membrane without having caused distinct disturbance. For this reason close attention should be given to pains in the right hypochondrium and epigastrium, and to fever and vomiting occurring one or two hours after the principal meal and disappearing after a few hours. For the appearance of these symptoms after partaking of food may be the initial symptom of fistula of the gall-bladder and intestine, and, therefore, significant in the diagnosis of a succeeding stenosis of the intestine due to gall-stones. In cases in which the stones find their way into the intestinal canal, the cause of intestinal occlusion is not the mechanical closure of the intestine, for the stones are rarely so large as wholly to obstruct its lumen, and because, at the autopsy, the calculi are found to be freely movable in the intestine. The occlusion is often due to a contraction of the organ around the stone, which is directly proven by those cases of stenosis of the intestine due to gall-stone in which laparotomy is performed. In the case of Rogner-Gusenthal, upon removal of the stone the spasmodic contraction of the intestine around it was seen. Stones which are impacted in the duodenum simulate pyloric stenosis. Frequent vomiting, dilatation of the stomach, sensitiveness of the right hypochondrium upon pressure, contraction of the musculature of the abdomen upon the right side, or friction sound in the gall-bladder region, will indicate the correct diagnosis, as well as, usually, the absence of melena and hematemesis, and, under some circumstances, the results of chemical and bacteriological examination of the gastric contents. If the stones are impacted in the vicinity of the ileo-cecal valve, appendicitis must be considered in the diagnosis. The localization of the pains in *McBurney's point,* rises in temperature, the age of the patient, etc., may lead to the true diagnosis. Stones in the sigmoid flexure have been mistaken for carcinoma. In very rare cases they may also lead to volvulus of the sigmoid. Diagnostically, it must be emphasized that in acute obstruction of the intestine from gall-stones the meteorism is usually slight; in volvulus of the sigmoid flexure, meteorism occurs early, increases rapidly, and reaches a high degree. In exceptional cases, the stones wandering in the intestine, if low down, and the abdominal walls flaccid, may be palpated by rectum in spite of meteorism and sensitiveness of the abdomen.

When the stone is lodged in the upper portion of the intestine, neoplasm is much more readily thought of than stone. In some cases of obstruction of the intestine due to gall-stones, the patients experience a sensation as though a firm body were moving in their intestines. Intestinal obstruction due to stone may be favored, that is, the stones may increase in size, by the apposition of fecal masses, such cases having been observed by Körte and Frerichs. Preceding colic, perhaps palpable tumor of the gall-bladder, the disappearance of the tumor with simultaneous symptoms of effusion of bile into the intestine, bilious vomiting, severe pains in the abdomen, and the symptoms of intestinal occlusion must all be considered in a diagnostic sense. The fact that stones have been previously found, the discharge of the same with the feces, as well as other symptoms of cholelithiasis, render the diagnosis positive. The occasional discharge of flatus, as well as the severity and rapid sequence of vomiting in intestinal obstruction due to gall-stone, in contrast to the longer intervals of vomiting in intestinal occlusion from other causes, may, in some cases, furnish a clue to the disease. The symptoms of occlusion may disappear and then recur. In the case of the aged a relapsing intestinal occlusion must suggest to us the possibility of a rupture of the stones into the intestine.

We see, therefore, that in the course of cholelithiasis the composition of the vomited material, the intensity of the act of vomiting, and the changes present in the lumen of the intestine may vary. First, as a rule, the contents of the stomach, subsequently those of the duodenum, and, finally, those of the jejunum and ileum are discharged. In the cases in which fecal vomiting occurs, the diagnosis of intestinal occlusion will not be difficult. If, however, the vomited material does not show fresh, unchanged bile, but bile which has already been acted upon by intestinal bacteria, which, therefore, originates from the lower portions of the intestine, its diagnostic significance as a premonitory sign of intestinal occlusion is obvious. In the early diagnosis of acute intestinal stenosis, the *vomiting of stercobilin* is, under some circumstances, an important finding, and I advise that an examination for stercobilin be made, particularly in those cases in which, after laparotomy, the question must be decided whether the acute appearance of so-called bilious vomiting is the consequence of peritonitis or of a change in the lumen of the intestine.

Hematemesis occurring in long-existing jaundice may be a sign of the hemorrhagic diathesis, of ulcerative rupture of the biliary passages into the stomach, of secondary gastric ulceration, or the consequence of thrombosis of the portal vein.

That lithogenous carcinoma of the choledochus and of the duodenum may sometimes run its course with hematemesis and melena is obvious. This is the more apparent since cases have been reported in which large stones situated in the choledochus, with and without accompanying jaundice, the stones being palpable through the abdominal walls, have been confounded with carcinomata of the pylorus and of the duodenum. The absence of hydrochloric acid in the vomited material, and the signs of gastrectasis, may in such cases, as is reported in literature, assist in the diagnosis. Stones in the common duct may change their position upon respiration, and even upon palpation may show slight movability, as is the case in carcinoma of the pylorus, in which circumstance, however, they differ from carcinoma of the head of the pancreas.

With an existing jaundice the condition of the gall-bladder may be of diagnostic importance, the latter, as has been mentioned, in carcinoma of the head of the pancreas being frequently felt as a tensely filled tumo and also sometimes visible, while in a tumor of the gall-bladder stone in the common duct are frequently absent in consequence of a precedin contraction of the gall-bladder. The motility of stones in the choledochus in contrast to carcinomatous obstructions therein, may also aid the surgeo during laparotomy.

Merkel describes a case of cholelithiasis which was very difficult t diagnosticate; it occurred in a woman, aged 51, who suffered from *di tressing, uncontrollable singultus*, intense abdominal pains which were n localized, with exacerbations after meals, and who had a pear-shaped, pai less tumor in the epigastrium. There was no jaundice, no vomiting; t stools were normal. The diagnosis wavered between tumor of the pylor and movable kidney. Operation showed, after incision of the tumor wh proved to be the adherent gall-bladder, a gall-stone weighing 15 gran which was an exact model of the gall-bladder that had contracted co pletely around it. Recovery.

The differential diagnosis between gastric carcinoma and cholelithia may also give rise to perplexity in cases in which the gastric carcino is ushered in with the signs of an acute attack of cholelithiasis, yet with producing a palpable tumor.

Such a case I presented to the Clinic on Nov. 24, 1893; it occur in a servant girl, aged 34, who reported that she had previously alw been well, but was suddenly attacked by colic-like pains in the right hy chondrium. These attacks steadily increased in frequency, and were followed by vomiting two or three hours after the ingestion of nourishment, loss of appetite, and, in the further course, jaundice. Free HCl was absent in the vomited material as well as lab ferment and lab zymogen; elongated lactic acid bacilli could not be demonstrated, but several eosinophilic cells were repeatedly found in the vomitus. Autopsy showed an infiltrated scirrhus of the pylorus with extreme stenosis of the latter, metastases of the mesenteric, retroperitoneal and periportal lymph-glands, compression of the common duct, and general jaundice.

In this case, which, on account of the sudden appearance of the symptoms, as well as by jaundice which was soon observed, resembled cholelithiasis, the *finding of eosinophilic cells in the vomited material* was of diagnostic importance, for the reason that these are significant of small cell infiltration of the interglandular tissue which consists of an accumulation of eosinophilic cells, as Hammerschlag demonstrated in gastric carcinoma.

The dilatation caused by cicatricial closure of the choledochus may reach an extreme degree, and may be palpated during life as a cyst-like tumor. Large stones impacted in the choledochus may compress the pancreatic duct and thus produce secondary infection of the pancreas and retention cysts; in complete closure of the pancreatic duct, by preventing the pancreatic juice from entering the intestine, they may give rise to extreme cachexia. The differential diagnosis of this latter form of cholelithiasis from carcinoma of the head of the pancreas may, therefore, be exceedingly difficult.

Morgagni reports a case in which the common duct was dilated to the size of a stomach and filled with stones. Such enormous dilatations of the common duct may readily be mistaken for pancreatic cysts, all the more so as, on account of the topographico-anatomical relations of the common duct to the pancreas and also from clinical experience, secondary affections of the pancreas (closure of the pancreatic duct, indurative pancreatitis, retention cysts) have been proven to depend upon cholelithiasis. The diagnosis of these conditions becomes even more puzzling in view of the fact that intense pains having the character of gall-stone colic occur also in pancreatic cysts, as in a case which I showed in the Clinic upon June 16, 1901, and in which I believed myself justified in making a diagnosis of pancreatic cyst. A farmer, aged 67, from Galicia, reported that for twenty-seven years he had been suffering from pains in the right epigastrium radiating directly to the lumbar region, usually occurring at night and lasting from one to two hours. These pains recurred at intervals of from one to two months up to the year 1900. There was never any jaundice. Preceding the attacks the urine was said to have been sometimes remarkably clear; during the attacks it was of a reddish color, without sediment or blood, and without stone. The bowels during the bile; the color of the feces in such cases depends principally upon the intensity of the intestinal peristalsis. If this is very intense, unaltered biliary coloring matter may be detected in the fluid stools. The feces are then yellow or green, and contain bilirubin and biliverdin. If the passage of the intestinal contents is so slow that the biliary coloring matter may be reduced, the feces become dark brown, sometimes tarry, and contain large amounts of stercobilin. The same is true in rupture of the gall-bladder or of the common duct into the duodenum. If, however, rupture occurs into the colon, it is evident that with the shorter road which the bile must traverse, and particularly with rapid peristalsis, the stools almost always contain undecomposed bile.

In the majority of cases the intestine is overflooded with accumulated bile, and *diarrhea* occurs. Ulcerating carcinomata of the biliary passages which perforate into the intestine may have the same effect. In a case in which, after the appearance of stools markedly stained with stercobilin without admixture of blood, and in which jaundice which had lasted a long time suddenly disappeared, the diagnosis of an ulcerative carcinoma of the common duct was made on account of the persisting marasmus of the patient. The autopsy showed an ulcerating villous cancer of the common duct. Accordingly, the composition of the stools may be utilized in the diagnosis of high or low perforation of the intestine.

Usually stubborn constipation is noted during the attack of colic. But there are also cases with diarrhea so profuse that, during the prevalence of a cholera epidemic, they may be mistaken for an attack of cholera. The diagnosis may also become difficult if poisoning, for example with arsenic, produce biliary colic; such cases have been observed. Stubborn diarrhea usually accompanies the forms of cholelithiasis which run their course in combination with acute septic infections, for example, endocarditis. But diarrhea, sometimes with hemorrhagic admixture, combined with anorexia, anemia and cachexia, may also be the principal manifestation of an insidious, but nevertheless severe, infection of the biliary passages.

Stones which have reached the intestine by perforation may, as is wel known, be lodged in the intestinal mucous membrane for months, but the may also, as is shown by cases reported in literature, cause stubborn diarrhea. Such diarrhea may be due to ulceration of the intestine, particularl in the region of the ileo-cecal valve. In cases in which there is a suspicio of intestinal ulceration, it is advisable to examine the feces for pyogen cocci, which, according to Gram, are positive (staphylococci and streptococci).

This aid to diagnosis, communicated to me by Kretz, has been su cessfully tested in our Clinic. As is well known, the majority of the norm intestinal flora of an adult consist of bacterium coli varieties, which a negative according to Gram, while cocci are present in the minority. I of bile above the stenosis, the variation in the color of the feces is explained similarly as the dribbling of urine in hypertrophy of the prostate gland. Analogous conditions may also occur in gall-stones which compress the common duct externally.

The patients sometimes first notice a whitish-gray color of the stools during the attack of colic, while they expressly emphasize that the urine a few days later resembles dark beer, and the skin becomes yellow. As regards the sequence of symptoms there are cases of gall-stone colic in which intestinal acholia precedes the jaundice and choluria.

Frequently this intermittent, ashy-gray color of the stools is combined with an acutely developing meteorism, which is the only objective manifestation of gall-stone colic.

A singer, aged 46, who was attacked by chills, pains radiating to the right shoulder, vomiting and diarrhea, reported that during the attack the abdomen became as tense as a drumhead, and the skin of the abdomen was glistening in appearance. The feces resembled those of an infant. There was no jaundice. This paradoxical phenomenon of *intestinal acholia without jaundice* requires special consideration, as it occurs particularly in those cases in which palpation of the gall-bladder reveals a distinct tumor. It is obvious in this instance that the gall-bladder acts as a reservoir, receiving the regurgitating bile from the over-filled biliary passages, which is naturally only possible when the cystic duct remains open.

The gall-bladder appears—at least in man and in many animals—to be a regulator of the intrabiliary pressure. This view is favored by the opinions of some authors who have observed a vicarious dilatation of the biliary passages after the closure of the gall-bladder and the cystic duct. Acholia of the feces without jaundice occurs also in fistula of the gall-bladder and abdominal walls after cholecystotomy; the explanation of this, according to Leichtenstern, may be that if a gall-bladder fistula exists biliary pressure cannot reach a degree necessary to overcome the tonus of the duodenal sphincter. Permanent intestinal acholia does not, therefore, justify the conclusion that an obstruction due to stone is present in the common duct, neither can it by any means be regarded as an indication for renewed operative interference.

According to Doyon, irritation of the central end of the vagus produces contraction of the gall-bladder and relaxation of the duodenal sphincter. It follows from this that a condition must exist in the living organism which necessitates the removal of this obstruction by antagonistic irritation of the peripheral branches of the pneumogastric nerve; therefore, this closed door may occasionally open, and become permeable for bile as well as for incarcerated stones in the common duct.

If, with the passage of stones into the intestine, the stasis of bile ceases, the retained bile pours into the intestine, and the acholic stools, or, at least, the stools deficient in bile, suddenly contain profuse amounts of bile; the color of the feces in such cases depends principally upon the intensity of the intestinal peristalsis. If this is very intense, unaltered biliary coloring matter may be detected in the fluid stools. The feces are then yellow or green, and contain bilirubin and biliverdin. If the passage of the intestinal contents is so slow that the biliary coloring matter may be reduced, the feces become dark brown, sometimes tarry, and contain large amounts of stercobilin. The same is true in rupture of the gall-bladder or of the common duct into the duodenum. If, however, rupture occurs into the colon, it is evident that with the shorter road which the bile must traverse, and particularly with rapid peristalsis, the stools almost always contain undecomposed bile.

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Melena in cholelithiasis is produced by the same causes that give rise to hematemesis, which have already been enumerated. In rare cases of wandering stone from the irritation of the mucous membrane, the feces may show hemorrhagic admixture, and this must be especially borne in mind in the differential diagnosis of neoplasms or intussusceptions of the intestine.

Special weight must be attached to the *examination of the stone passed in the feces,* which is simultaneously a *corpus delicti* of cholelithiasis. Negative examination of the stool, however, does not permit the conclusion that no stones have been passed; for, according to Naunyn, stones of soft consistence may be disintegrated in the intestine. As we already know, the gall-bladder is the point of formation of stones. The stones which have wandered out of the gall-bladder, which have traversed the cystic duct, may, instead of taking their ordinary course into the wider common duct to the point of the duodenal papilla, move in a retrogressive direction, may be forced upward into the branches of the hepatic duct, and thus produce a cholelithiasis intrahepatica with all its dangerous tendency to biliary abscesses. This is the manner in which *cholelithiasis intrahepatica* of exogenous origin arises.

The development of stones in the branches of the hepatic duct may also be autochthonous in this locality, and without generating particular disturbance, they may find their way into the wider common duct. However, it is quite possible that the stones which have developed in the hepatic duct, instead of entering the common duct with the flow of bile, find their way to the gall-bladder where they then enlarge. We see, therefore, that stones in the choledochus may be of different origin, from the hepatic duct, from the gall-bladder, arising autochthonously by crystallization around cholesterin, and, finally, by the perforation of the gall-bladder and its diverticulum into the common duct.

Kehr has observed cases in which, in spite of large stones being impacted in the neck of the gall-bladder, small stones were found in the common duct. Stones may be situated in the cystic duct, and also simultaneously in the hepatic duct. After the extraction of stones from the gall-bladder, stones in the hepatic duct may move downward, which is of importance in considering the question of cholecystotomy or cholecystectomy.

The seat of the stone in some cases corresponds to a local area of pain, which persists even when narcotics are administered to the patient, as, for example, injections of morphin.

The seat of the stone may sometimes be revealed by the sequence of the symptoms. In the overwhelming majority of cases preceding colic with local signs in the gall-bladder, a tumor, a collection of fluid, pain, then the disappearance of these symptoms, and an outbreak of permanent jaundice indicate that the seat of the stone is the common duct.

The fate of the gall-stones in the intestine varies. They may break up in their passage through the intestine, they may do much damage, small stones may be arrested in the appendix, and large ones in the ileo-cecal valve. Stones which have formed in the gall-bladder may, by producing a diverticulum, directly penetrate the substance of the liver, by perforation of the posterior wall of the gall-bladder they may produce a paranephritic abscess, after rupture they may appear in the lumbar region; in perforating downward they may also find their way into the urinary bladder, and, after long wanderings, we may be surprised to find them even in distant parts of the body; for instance, as in a case of Porges, who extracted a quantity of gall-stones from a fistula in the thigh of a woman.

In examining the stones passed in the feces, above all their size is to be noted. The question whether large stones can pass the conical, terminal portion of the ostium duodenale is variously answered by pathological anatomists. In opposition to the view of Rokitansky, who believes it possible, on account of the great power of dilatation of the common duct, that even stones the size of a hen's egg may pass the ostium duodenale choledochi, Roth maintains that such seeming dilatations are really typical perforations of the common duct, these being usually situated from 5 to 10 mm. above the ostium duodenale, and in these perforations the upper portion of the portio intestinalis ductus choledochi communicates with the descending part of the duodenum. The greatest dilatation of the ostium duodenale produced by gall-stones had, according to Roth, the diameter of a medium-sized pea. He does not believe it likely, therefore, that stones of greater dimensions reach the intestine naturally.

At the bedside, the passage of large stones after preceding grave symptoms is a sign of perforation, and with renewed colic forms, as Riede says, a contra indication to operation, as it is obviously possible that mor stones may pass through the perforation. If at first small stones, and the somewhat larger ones pass, we are justified in concluding that the commo gall duct is dilated.

After a single passage of stones the biliary passages remain dilate and this facilitates the later passage of succeeding stones so that, if smal they may easily be discharged in the feces.

The passage of equally large stones which are faceted may—*cæter paribus*—form a contra indication to operation, inasmuch as we assum that the stones remaining in the gall-bladder may pass in the same way as did their predecessors. The repetition of attacks without the passage of stones awakens the suspicion that large stones are also present in the gall-bladder. According to Riedel, the small stones which hide themselves in the common gall-duct, and occasionally slip into the hepatic duct, cause anxiety to the surgeon. A stone that possesses only one facet permits the conclusion that it is the first or the last of a series.

As the nucleus of stones, intestinal parasites, portions of the distoma hepaticum, and once the stone of a plum have been found.

Gall-stones are composed chiefly of cholesterin with a slight admixture of bilirubin calcium. Carbonate and phosphate stones must be regarded as pancreatic stones, but are not invariably so, as is proven by the two cases published by Eichhorst, who, in a case of mucous colic, found a profusion of small granular stones of from ½ to 2 mm. in diameter which consisted of calcium carbonate. The seed of fruit (pears), the vertebra of birds, and chalk introduced for medical purposes must also be considered. It follows from this that the chemical, microscopical (for distoma), and perhaps also the bacteriologic (for typhoid bacilli) investigation of stones that have been passed may furnish valuable information regarding their origin, their past, and, eventually, also their etiology.

The amount of *urine* in cholelithiasis is either normal, diminished, or increased. A few cases have been observed of polyuria with bulimia and thirst but without glycosuria. In some instances, at the onset of the attack the excretion of a transparent urine in increased amount is reported (urina spastica). Retention of urine also occurs, particularly after the internal or subcutaneous administration of morphin. Choluria in jaundice is a well-known symptom. Conspicuous choluria without jaundice must lead us to suspect a perforation of the gall-bladder into the urinary bladder. Transitory albuminuria from hyperemia of the kidneys arises during the attack, and disappears after its cessation. Albuminuria with blood and casts occurs in cholangitis in consequence of infectious nephritis. Similar conditions are observed in occlusion of the common duct and chronic jaundice. Glycosuria, peptonuria, and acetonuria during the attack have been reported by different authors.

The excretion of sodium urate and uric acid as a *sedimentum lateritium* is not of rare occurrence.

In one case leucin and even tyrosin crystals were found for weeks. The excretion of indican is usually normal; sometimes, however, it has been found increased.

A woman, aged 38, the widow of a letter carrier, was admitted to the clinic with a diagnosis of cholelithiasis; during the attack, a conspicuous increase in indican was found. Operation revealed a contracted gall-bladder largely adherent to the omentum, which was tensely filled with stones. It may be that the adhesion of the gall-bladder to the omentum calculosa and nothing abnormal in the umbilical hernia. Removal of the stone was followed by recovery.

Large stones, which by perforation of the common duct or the gall-bladder reach the intestine, may produce symptoms of occlusion, and either soon after their entrance or a long time afterward, even weeks, months or years, severe symptoms of intestinal stenosis may appear. After prolonged retention in the intestine owing to peristaltic and antiperistaltic movements, they may become fixed in some portion by spastic contraction, either in the duodenum, jejunum or ileum, usually in the region of the ileo-cecal valve. Large stones may also reach the cecum and the large intestine after dilatation or ulceration of Bauhin's valve (ileo-cecal). In this manner even stones the size of a hen's egg may be discharged with the feces. In other cases, they lead to ulceration or perforation of the intestine with all their consequences. Rupture may occur directly into the peritoneal cavity and into cavities formed by preceding inflammation, and thus circumscribed abscesses develop. This shows how valuable for correct diagnosis are many obscure cases of intestinal occlusion or ulceration in the ileo-cecal region, and how important is an accurate history which may eventually point to a preceding cholelithiasis. This is all the more true since the stones which have reached the intestine by perforation may have been present for months and even for years in the intestinal mucous membrane without having caused distinct disturbance. For this reason close attention should be given to pains in the right hypochondrium and epigastrium, and to fever and vomiting occurring one or two hours after the principal meal and disappearing after a few hours. For the appearance of these symptoms after partaking of food may be the initial symptom of fistula of the gall-bladder and intestine, and, therefore, significant in the diagnosis of a succeeding stenosis of the intestine due to gall-stones. In cases in which the stones find their way into the intestinal canal, the cause of intestinal occlusion is not the mechanical closure of the intestine, for the stones are rarely so large as wholly to obstruct its lumen, and because, at the autopsy, the calculi are found to be freely movable in the intestine. The occlusion is often due to a contraction of the organ around the stone, which is directly proven by those cases of stenosis of the intestine due to gall-stone in which laparotomy is performed. In the case of Rogner-Gusenthal, upon removal of the stone the spasmodic contraction of the intestine around it was seen. Stones which are impacted in the duodenum simulate pyloric stenosis. Frequent vomiting, dilatation of the stomach, sensitiveness of the right hypochondrium upon pressure, contraction of the musculature of the abdomen upon the right side, or friction sound in the gall-bladder region, will indicate the correct diagnosis, as well as, usually, the absence of melena and hematemesis, and, under some circumstances, the results of chemical and bacteriological examination of the gastric contents. If the stones are impacted in the vicinity of the ileo-cecal valve, appendicitis must be considered in the diagnosis. The localization of the pains in *McBurney's point*, rises in temperature, the age of the patient, etc., may lead to the true diagnosis. Stones in the sigmoid flexure have been mistaken for carcinoma. In very rare cases they may also lead to volvulus of the sigmoid. Diagnostically, it must be emphasized that in acute obstruction of the intestine from gall-stones the meteorism is usually slight; in volvulus of the sigmoid flexure, meteorism occurs early, increases rapidly, and reaches a high degree. In exceptional cases, the stones wandering in the intestine, if low down, and the abdominal walls flaccid, may be palpated by rectum in spite of meteorism and sensitiveness of the abdomen.

When the stone is lodged in the upper portion of the intestine, neoplasm is much more readily thought of than stone. In some cases of obstruction of the intestine due to gall-stones, the patients experience a sensation as though a firm body were moving in their intestines. Intestinal obstruction due to stone may be favored, that is, the stones may increase in size, by the apposition of fecal masses, such cases having been observed by Körte and Frerichs. Preceding colic, perhaps palpable tumor of the gall-bladder, the disappearance of the tumor with simultaneous symptoms of effusion of bile into the intestine, bilious vomiting, severe pains in the abdomen, and the symptoms of intestinal occlusion must all be considered in a diagnostic sense. The fact that stones have been previously found, the discharge of the same with the feces, as well as other symptoms of cholelithiasis, render the diagnosis positive. The occasional discharge of flatus, as well as the severity and rapid sequence of vomiting in intestinal obstruction due to gall-stone, in contrast to the longer intervals of vomiting in intestinal occlusion from other causes, may, in some cases, furnish a clue to the disease. The symptoms of occlusion may disappear and then recur. In the case of the aged a relapsing intestinal occlusion must suggest to us the possibility of a rupture of the stones into the intestine.

We see, therefore, that in the course of cholelithiasis the composition of the vomited material, the intensity of the act of vomiting, and the changes present in the lumen of the intestine may vary. First, as a rule, the contents of the stomach, subsequently those of the duodenum, and, finally, those of the jejunum and ileum are discharged. In the cases in which fecal vomiting occurs, the diagnosis of intestinal occlusion will not be difficult. If, however, the vomited material does not show fresh, unchanged bile, but bile which has already been acted upon by intestinal bacteria, which, therefore, originates from the lower portions of the intestine, its diagnostic significance as a premonitory sign of intestinal occlusion is obvious. In the early diagnosis of acute intestinal stenosis, the *vomiting of stercobilin* is, under some circumstances, an important finding, and I advise that an examination for stercobilin be made, particularly in and suppurative inflammation of the biliary passages and the portal vein, or empyemia due to ordinary pus cocci, or ichorous empyemata with fetid pus in which, as in a case of Zuber and Leroboulet, anaërobic microbes were found. The dangers from obstruction of the common duct, cholemia, jaundice with hemorrhage appearing after a short time, and secondary acute hepatic atrophy, are no less threatening. The more chronic consequences and complications are biliary induration and cirrhosis, chronic suppuration of the peritoneum, adhesions with stenosis of the duodenum and of the remaining parts of the intestine by giving rise to gastro-intestinal disturbances, in some cases increased by morphinism, and, finally, cancer of the gall-bladder and biliary passages, all of which may cause death.

TREATMENT

On account of the many aspects of the disease and according to the stage in which we find it, the objects of treatment are manifold. At one time the colic most requires our therapy, in other cases closure of the cystic duct from stone, of the hepatic and common ducts, at other times the infection of the gall passages and peritoneal processes, or intestinal occlusion, and, finally, it must be our aim to prevent the formation of new gall-stones. In *the treatment of the paroxysms of pain,* opium and its preparations remain the sovereign remedy. Belladonna, atropin, chloroform and ether are advised for the same purpose. By some authorities the use of oil and glycerin is advocated. Hot poultices, hot cataplasms, in other cases the ice-bag, leeches, and protracted warm baths are the therapeutic measures with which to combat the pain.

In regard to *diet* there is still great diversity of opinion. According to experience, it appears that a mixed diet most thoroughly meets the indications. A plentiful intake of fluid, particularly the alkaline, chlorin-containing and saline mineral waters is favored by practice.

For the relief of the gastric and intestinal disturbances, as well as the jaundice, Fleiner has advised *gastric lavage,* which he believes to be more efficacious than mineral spring cures, but in regard to which I have no personal experience. Fleiner believes that by the influx of water into the small intestine, as occurs in gastric lavage, there is resorption of the same in the portal veins, and thus the liver and the entire body are washed out. Lately, systematic washings with silver nitrate solution at a temperature from 122° to 131° F. (50° to 55° C.) have been advised by Ehrlich.

These energetic procedures may, however, change a latent cholelithiasis into the actual stage of wandering of the stone. Stones which are latent or which produce only slight difficulty, are to be treated as a ***noli m*** *tangere,* or, more correctly, "*quieta non movere.*" As the danger consist largely in the wandering of the stones, and this wandering is sometime induced by a Carlsbad cure, we must carefully consider whether it is advisable to send patients with slight symptoms to Carlsbad; in regard to the value of this old and popular remedy, a Carlsbad mineral-spring treatment, there is far from unanimity of opinion. According to Riedel, Carlsbad water alleviates the pain of cholecystitis by its purgative effect, by reducing the hyperemia of the inflamed mucous membrane of the intestine, but it simultaneously retards the expulsion of the stones. If the stones are passed while in Carlsbad, this does not occur because of, but in spite of, Carlsbad.

[In the treatment of cholelithiasis with infection of the biliary apparatus and in cholangitis without stones, many clinicians have found salicylic acid of value. Salicylic acid is excreted by the liver cells or at any rate, when ingested, it may be found in the bile. It is an antiseptic. It has been used sufficiently to prove its usefulness in infectious conditions of the biliary apparatus with and without gall-stones. It may be used alone or combined with alkalies and laxatives. I have used with good results the following:

℞ Sodii salicylatis	1.0
Sodii phosphatis Gran.	2.0
Sodii sulphatis Exsic.	6.0

Misce; signa. One teaspoonful in hot water one-half hour before meals, three or four times a day.

Or one may give phenol salicylas 0.3 in pill three or four times a day, and a sufficient dose of Carlsbad salts or magnesii sulphas in hot water on the fasting stomach to act as a brisk laxative.

Oleate of soda is also excreted by the liver and undoubtedly renders the bile more fluid. It may be given as a soda soap in the form of pills or in the form of ol. olivae in sufficient dose to act also as a laxative. Free intestinal action is of unquestioned value in cholelithiasis with infection. The "internal drainage" established by this method probably explains the frequent relief from cholangitis with gall-stones.

Gall-stone disease must be recognized as a surgical disease. The physician, with benefit, may utilize prophylactic measures, may reduce the accompanying *pain, vomiting* and *infection,* but only the surgeon surely may remove the calculus and correct the consequent morbid anatomy (adhesions, etc.). The danger of cholangitis, hepatic abscess, perigastric adhesions, pancreatitis, etc., occurring as a result of gall-stones is so great that even the most conservative physician may well hesitate to take the responsibility of non-surgical treatment.—Ed.]

The greatest difference of opinion prevails in regard to the question whether *early operation or conservative treatment* is indicated. In the cases in which the course is unusually protracted and painful and there is loss of strength, and in which repeated mineral spring treatments are ineffectual, in those cases in which an empyema of the gall-bladder threatens perforation, or an irreducible stone obstruction of the common duct is present, no internal clinician will veto an operation. It is different, however, in regard to our justification for an operation as early as possible, and it would be interesting to know whether the surgeon who is convinced of the wisdom of radical operation, and who himself has gall-stones, would not prefer to give Carlsbad a trial before consenting to an operation. Certainly general rules cannot be laid down.

The method of operation is indicated after minute weighing of all the factors which are decisive for the topical diagnosis in a concrete case. Where the diagnosis has been incorrect, for example, when cholelithiasis has been diagnosticated as wandering kidney, and the surgeon, pursuing this incorrect diagnosis, attacks the supposed wandering kidney posteriorly, and finds a stone in the cystic duct, the uselessness of operative procedure in accordance with a laid-out program is apparent.

The specter fear of the serious consequences of cholelithiasis, which the surgeons love to oppose to a conservative treatment, is much weakened by contrary considerations. In the first place, quite a series of even desperate cases have been known which were nevertheless cured in consequence of perforation. Moreover, secondary stone formation in the tied stump of the cystic duct and in the biliary passages remains a sword of Damocles even after a successful operation, and, finally, in the cicatrix of operation a carcinoma may develop similarly as in the cicatrix of ulcer.

The surgery of the biliary passages is yet too recent to permit a conclusive opinion in regard to the *permanent results* of operation, and we are forced to agree with Wölfler, who says that surgery of the biliary passages has for several years presented no reliable statistics, and holds that those operated upon should be observed for at least a decade.

Since cholesterin stones, which alone come into question here, may develop in any part of the biliary passages in which there is mucous membrane epithelium, we can, in fact, speak of a radical prophylactic gall-stone operation only when it is possible to eradicate the entire cholesterin-forming mucous membrane epithelia.

ETIOLOGY

The treatment of cholelithiasis, particularly its prophylaxis, rests upon an uncertain foundation, principally for the reason that accurate knowledge of the near and remote causes of gall-stone formation is completely lacking. At present two theories chiefly dominate the *etiology* of gall-stones, the humoral and the infectious. The champions of the one regard cholelithiasis as a constitutional affection, a link in the chain of that diathesis whose family tree is arthritis. They base their opinions chiefly upon the occurrence of gall-stones in families in which, simultaneously, obesity, gout, diabetes, hemorrhoids, eczema, increased uric acid excretion, and oxaluria, etc., are observed. The genesis of infection by means of the circulation or from the intestine is constantly gaining ground. This is based particularly upon the finding of the bacterium coli, and even upon the typhoid bacillus as the nucleus of gall-stones, as well as upon the similarity and the length of time the stones have been in the gall-bladder, which, according to Naunyn, may best be explained by a single bacterial infection.

In the humoral theory, it would be necessary to assume that the physiologic composition of the bile, that is, the relation between sodium-containing bile and cholesterin, has been altered in a pathological manner, and that the liver, to begin with, secretes a pathologically altered bile which readily forms a sediment, takes the shape of a ball, and grows into a clumpy stone. In the case of man, cholesterin is chiefly important since it forms the principal mass of gall-stones, while bilirubin calcium is to be looked upon only as a cement. The fact that cholesterin forms the chemical base of most human gall-stones is in contrast to the stones of the herbivora, which consist principally of bilirubin calcium, so that the gall-stones of oxen form a suitable material from which to prepare pure bilirubin in chemical laboratories. The influence of food and drinking-water, which is maintained by several authorities, would, therefore, be operative only in the case of bilirubin stones. Experienced cattle-raisers maintain that gall-stones are observed with surprising frequency in cattle and swine coming from regions where the meadows contain saltpeter, and this frequently permits a direct conclusion as to the regions from which the animals come. In the etiology of human stones, their rare occurrence with long-standing inspissated bile by the occlusion of the choledochus (catarrhal jaundice) is noteworthy, and the view is obvious that the causal factor of gall-stone formation is to be sought either in the blood from which the liver cells prepare bile, or in the liver cells themselves, which furnish a pathological secretion.

The chemical examination of the blood in cholesterin anemia, as well as in regard to its diminution of alkalinity and the deficiency of sodium-containing biliary acids, has been negative up to the present time. No investigations have as yet been made of hemoglobin in regard to its solubility, its power of crystallization, its faculty of coagulation, or its hygroscopic relation. It is possible that the hemoglobin varieties of different individuals are not identical, and that the hemoglobin of gall-stone patients is different from that of a normal person; such individuals may, perhaps, secrete a bile which crystallizes very readily, similarly as the patient with the urate or oxalate diathesis, who, without showing symptoms, intermittently excretes uric acid or calcium oxalate crystals in his urine.

If, however, to this congenital or acquired abnormality in the quantitative composition of the bile, we add mechanical stasis, corset liver, obesity, pregnancy, large abdominal tumors, hepatoptosis, hindrance of the diaphragm action, senile atrophy of the musculature of the gall-bladder, a sedentary mode of life, infrequent meals, or bacterial infection, it is easy to conceive that, similarly as when uric acid stones are present, here also an agglomeration of cholesterin precipitate may result in gall-stones. In this sense, cholelithiasis may be regarded as a diathesis of the tissues.

By analogy, we might suppose that not the blood, but the liver cells themselves are affected, and that in their decomposition they furnish cholesterin, the possibility of which is admitted by Naunyn. In opposition to this is my personal experience, according to which gall-stones do not occur in alcoholic cirrhosis. It therefore follows that the destroyed liver cell, damaged by alcohol, furnishes bile which is not a suitable material for gall-stone formation although in its quantity, external appearance, color and consistence it does not decidedly deviate from the normal. Reports of microscopic and chemical examinations of bile in such cases are not at hand, and, so far as I know, have not been made.

Some of the factors are also in opposition to the theory of an infectious origin. In a general affection of the biliary passages the excretion of pathogenic bacteria is frequently observed. The presence of typhoid bacilli in the gall-bladder as the only bacterial flora—which makes an invasion by way of the intestine very unlikely, but which, on the other hand, favors the introduction by way of the circulation—is an ordinary finding in the course of enteric fever. Bacteriemia is frequent in ulcerative tuberculosis, while gall-stones in tuberculosis, as well as after a general bacterial infection, are seldom observed. Even in the cholelithiasis which follows these maladies an absolute relation between the diseases *cannot* be proven.

To explain acute serous, rapidly retrogressing cholecystitis, which develops a sterile, transparent dropsy of the gall-bladder by an infection from the bacterium coli in which the latter organism has been killed, appears to me somewhat far-fetched, if we take into consideration that infections from the bacterium coli, as, for example, in cystitis or in pyelitis, are by no means of such short duration. Why should the gall-bladder in particular be so slightly injured by the bacterium coli, especially since it has been proven that bile is an excellent culture medium for this bacterium? In other mucous membranes we are familiar with acute swelling and increased secretion which cannot be regarded either as inflammation or as infection. I refer here particularly to coryza vasomotoria and to bronchial asthma, which cannot be regarded as infectious mucous membrane inflammations, either from a bacteriologic or clinical standpoint.

Some forms of cholelithiasis marked by the sudden disappearance of decided and painful tumors of the gall-bladder with all of their accompanying symptoms remind us of the turbulent catarrhal symptoms with which bronchial asthma runs its course, a true "parturiunt montes" which, as Kehr remarked of infectious cholecystitis, "often resembles a fire of straw that blazes up but once and is rapidly extinguished." I presume that at least many forms of this gall-stone colic which have been explained as abortive infections belong to the group of *neuroses of secretion*, and are, therefore, in fact, *catarrhal asthma of the biliary passages*. For the gall-stone quiescent in the gall-bladder only occasionally assumes the rôle of a provocative agent for the development of colic, as, for example, nasal polypi in the development of bronchial asthma. Besides the abnormal secretion which is peculiar to asthma, spasm of the bronchial muscle is a prominent symptom; the accompanying fulminant nervous signs are analogous to those of gall-stone colic in which the secretion stored in the gall-bladder forms a motive power for the wandering stone. In acute serous dropsy of the gall-bladder, which at first is sterile but which by irritation of the foreign body and secondary bacterial infection may lead to cholecystitis, the effect of the operative removal of stones may be compared to that of the extraction of nasal polypi in asthma. Just as infectious bronchitis and lobular pneumonia may develop in the course of bronchial asthma, so secondary infections of the biliary passages play an important rôle in the pathology of cholelithiasis. Although it is evident that, in most cases, these infections merely change the latent to the actual stage of cholelithiasis, I am inclined to believe that quite a number of cases of gall-stone colic are neither inflammatory nor infectious. The capricious behavior of some cases, with a latency sometimes lasting for years, an eruptive appearance after psychical emotion and a sudden disappearance, the rapid sequence of relapses, the long intervals of health—these peculiarities are not readily explained by the assumption of a transitory infection, and therefore I regard these cases which run their course with acute dropsy of the gall-bladder as neuroses of secretion, parallel to those observed in catarrhal asthma.

Rokitansky[1] points out that pure cholesterin stones may be excreted without bile in consequence of the pathological secretion of transparent synovial-like fluid in dropsy of the gall-bladder, in complete occlusion of the cystic duct. This opinion was first clearly formulated by Naunyn who also found stones in a diverticulum, "surrounded by mucous membrane," which for a long time had precluded the presence of bile. Cholesterin is, however, produced by the mucous membrane itself, while bilirubin stones are formed only where bile is found.

These findings of Rokitansky and Naunyn alone favor the assumption that calculus formation is a local product of the epithelia of the gall-bladder, that is, of the epithelia of the biliary passages, and that the bile, as such, has no action in the original formation of the stones. Naunyn

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[1] "Handbuch der speciellen pathologischen Anatomie," 1842–1844.

ACUTE DIFFUSE PERITONITIS, APPENDICITIS, AND PERITYPHLITIS

I. DIFFUSE AND CIRCUMSCRIBED PERITONITIS

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ACUTE DIFFUSE PERITONITIS

As an introduction to the discussion of our present views of acute peritonitis I will relate the following clinical history:

Case I.—A previously healthy merchant, aged 31, was taken ill after a few days of vague, dull pain in the right side of the abdomen which he had disregarded, and upon the 20th of October, about midday, he was seized with very severe pain in the right lower abdominal region which compelled him to seek his bed; soon afterward he had chilly sensations which increased to marked chills; there was also nausea, eructation and vomiting, first of food and then of bilious mucus; a little later tenesmus appeared, the patient first voiding small, compact feces, followed by scant, thin dejecta. Within a few hours the abdomen had become tympanitic, the pains continued with exacerbations upon motion, after eructations, and on talking; the entire abdomen was very sensitive. Strangury with the frequent discharge of scant urine was observed.

Toward evening the physician found the patient extremely ill, immovable in the active dorsal decubitus, with an anxious facial expression, reddened cheeks, cautious, superficial respiration with a low, hushed voice; he complained of continuous, also occasionally of marked tearing and contracting, pains in the entire abdomen, most severe upon the right side low down; the temperature was 103.2° F., the pulse was 112, full, somewhat tense, regular and even.

The lips were dry, the tongue markedly coated; *fœtor ex ore* was present; painful eructations were frequent, also singultus, complete anorexia and extreme thirst. The respirations were superficial, quite rapid, and purely thoracic; the diaphragm was slightly raised; the pulmonary liver border was in the right mammillary line at the lower border of the fifth rib; upon anterior examination the thoracic organs appeared normal; the examination of the back was not then undertaken.

The entire abdomen was uniformly tympanitic, everywhere very sensitive to the slightest pressure, but more so upon the right side than upon the left. There was also pain upon pressure in the lumbar region.

Signs of abdominal respiration were absent. Careful palpation showed a uniform, drum-like resistance, otherwise nothing abnormal. The percussion note over the abdomen upon light tapping (and only this could be borne) revealed no decided difference, and nowhere any dulness; upon prolonged continued auscultation, high-pitched intestinal murmurs were here and there heard.

Retraction of the thighs produced diffuse abdominal pain, more marked upon the right side than upon the left; careful examination of the hernial rings gave a negative result.

Upon careful digital exploration per rectum in the dorsal decubitus, nothing abnormal was noted except pain in the floor of the pelvis; the rectum was empty.

Since morning neither feces nor flatus had been passed; the patient complained of strangury which, however, he rarely attempted to relieve because he feared to aggravate the pain which shot downward and radiated into the urethra. The urine was of high color, clear, and contained a trace of albumin and large amounts of indican.

The physician in charge of the case diagnosticated acute, diffuse peritonitis, the origin of which was not quite clear; very likely it was in the appendix. He ordered absolute rest, that the urine and feces be voided in the recumbent posture; that, for the present, only small quantities of ice be taken by the mouth; that two bags filled with ice be applied to the abdomen, and be suspended from a hook if they could not be borne directly upon the abdomen. Furthermore, at first every two hours, later somewhat less frequently, 0.03 of opium purum in powder form was to be taken in a little water.

In the following forty-eight hours, with irregular variations and a slight tendency to rise, the temperature ranged between 102.2° and 105.3° F. The pulse became more frequent but remained strong and uniform; the respirations were unaltered in character but increased in frequency to 48. The patient, unless under the influence of opium, was sleepless, his mind was clear, and he gave us the impression of being extremely ill, although not in collapse. The pains, eructation and vomiting were decidedly relieved by the opium; but ice-bags for a time were not well borne and cold Priessnitz compresses were substituted. Vomiting was rare, was invariably bilious and coarse-grained; neither feces nor flatus were discharged; the urine was as before, the diazo-reaction negative.

Distention of the abdomen and the area of diffuse resistance increased; sensitiveness to touch appeared to be dulled by the opium; in the ileo-cecal region, however, it was constantly severe and lancinating. The liver dulness below decreased; the pulmonary liver border extended to the upper border of the fifth rib; on the right side of the abdomen between the navel and the anterior superior spine of the ilium a circumscribed slight dulness was observed. There was great nausea and burning thirst.

Diagnosis: Acute diffuse appendicular peritonitis, probably also perforation; circumscribed perityphlitic abscess.

Operation was considered but not performed. Removal to the hospital for the purpose of an operation was absolutely declined by the patient.

I saw him upon the following day, the fourth of the disease. In general the severity of the clinical picture had increased, especially some of the individual symptoms: Severe, markedly febrile general condition; temperature about 104° F. and showing moderate remissions; pulse 120 to 136, moderately full, regular. There was insomnia with occasional opium slumber; otherwise the mind was clear but anxious. The tongue was thickly coated, the lips were dry, there was tormenting thirst. The cheeks were red. The patient maintained the dorsal decubitus with feebly flexed legs and hushed voice; the hands moved but slightly and trembled. Occasionally there were spontaneous attacks of severe, tearing, abdominal pain, starting posteriorly in the lower right side. The abdomen was very tympanitic and tense, and could scarcely be touched; nevertheless, it was possible to determine upon the right side low down an area of dulness about the size of a hand with increased resistance; otherwise the note was tympanitic upon percussion. The diaphragm was raised; except for a small zone liver dulness was absent. Now and then there was grass-green vomitus which, the last time, contained a few brownish granules and had a fecal odor. Urine unchanged; micturition very painful; no feces.

Opium at first decidedly influenced the condition; the patient took daily 0.5 to 1.8, and since yesterday morphin subcutaneously 0.02 at a dose. Ice-bags were not well borne, and Priessnitz compresses were used continuously. The intake of food was reduced to almost nothing.

With a sharply circumscribed perityphlitic abscess there could be no doubt of the diagnosis of diffuse peritonitis nor of the indication for operation on account of the long continuance of the severe symptoms. But neither this proposition nor that of an exploratory laparotomy, the result of which might have induced the patient to yield, was accepted.

Morphin was ordered subcutaneously, Priessnitz compresses to the abdomen, pellets of ice and meat jelly by mouth; eventually gastric lavage.

Upon the sixth day of the disease the picture changed: The complexion became sallow, the face elongated, the eyes hollow; the pulse was 140, small, but quite regular; the temperature was 101.3° F.; there was clammy perspiration and a cool skin, the hands were cold; frequently slight eructations occurred and, now and then, ineffectual or mild paroxysms of vomiting of a greenish yellow material with a slight fecal odor. The mind was clear; there was little pain. The abdomen became somewhat softer, much less painful, and was readily palpated and percussed; there was a distinct resistance about the size of a hand, quite firm, and not fluctuating, and accompanied by marked dulness, around McBurney's point and downward, and only in this region severe stabbing pain; in other areas no dulness.

The symptoms were those of moderately severe *peritoneal collapse*; the prognosis was very grave although not positively hopeless. Treatment: Small quantities of alcohol, to be followed by camphor.

This condition lasted nearly twenty-four hours; then a very large and hard stool, followed by a thin one of hemorrhagico-purulent character was discharged and simultaneously a decided change took place: The appearance and pulse improved; the abdomen became softer with the exception of the marked resistance upon the right side low down, and the fever slightly remittent, its maximum 101.1° F. Vomiting did not recur; the patient moved about somewhat in bed and slept several hours in a half-lateral posture. Meat jelly and cold beef tea were swallowed.

Upon the next day there were several hemorrhagico-purulent stools, the urine was profuse and voided without pain. Nevertheless, a firm, flat resistance was still felt in the lower right side and upon pressure there was lancinating pain; no fever.

There could be no doubt that the perityphlitic abscess had ruptured into the intestine, and that in consequence of this the diffuse peritonitis had at once been relieved.

Treatment: Warm, followed by hot, flax-seed poultices; rest; freshly expressed meat juice or beef tea, in all 200 grams; thin gruel made with milk, 200 grams; wine, 100 grams in twenty-four hours, small portions to be taken every two hours; no drugs.

This improvement continued for several days and even became more marked. The abdomen returned to the norm with the exception of the ileo-cecal region; there was a small stool daily without recognizable pus; no fever.

Upon the *twelfth day of the disease* vomiting suddenly recurred, with severe, diffuse abdominal pain, marked meteorism, and fever to about 102.2° F.; the symptoms increased in severity, and changed during the night. The next morning I found the patient in collapse, his temperature 97.3° F., pulse 160, thready, uneven; conspicuous facies hippocratica; no pain; a slightly comatose condition, moderate meteorism, no movement of the bowels. Stimulants were without effect; subcutaneous saline infusion revived the patient but only for a short time, and death occurred the following morning upon the fourteenth day of the disease.

Autopsy: Normal condition of the serosa above the omentum; the appendix surrounded by adhesions embedded in fecal pus, gangrenous toward its terminal portion, and showing perforation; fecal calculus in the pus; appendix movable toward the cecum. Agglutinated point of rupture at the median periphery of the cecum near the ileo-cecal valve. The perityphlitic pus appeared to be sacculated by adherent intestinal coils, but beyond the adhesions in the free abdominal cavity below the omentum there was diffuse, fresh, fibrinous peritonitis and distributed here and there small quantities of thin, putrid pus (many bacteria, large quantities of streptococci and coli bacilli). The peritoneum was injected, of a delicate rose-red color, here and there covered with fine, mucus-like pseudo-membranes. Heart flabby.

This clinical history is in every respect typical and instructive.

It shows us the origin of peritonitis which is by far the most common: in a diseased appendix. At the autopsy this was found necrotic and perforated. It is questionable whether the perforation existed from the onset of the disease; it is possible that at first an ulcer extending to the serosa caused an infection of the peritoneum; at all events this occurred acutely, and produced the sharply defined disease. The clinical abdominal symptoms in the first period of the malady pointed to the fact that at the onset there had been a diffuse inflammation of the peritoneum, and that later by the adhesions to the appendix which were found at the autopsy an early encapsulation of pus had taken place in the ileo-cecal region; this produced a purulent softening in the wall of the cecum and led to the favorable rupture of pus into the intestine and to an immediate amelioration of the acute peritonitis. The point of rupture, however, then closed, and partly to this circumstance, partly perhaps to the action of fresh infectious and toxic material, perhaps only to the perforation of the appendix, may be ascribed the exacerbation of the peritonitis, that is, a renewed attack which caused the death of the patient. In regard to the fulminant symptoms at the onset of the disease, however, it is more likely that even then perforation had already occurred, and that the final and fatal exacerbation was in consequence of adhesions formed in the first period which were powerless to resist the entrance of organisms producing inflammation. The pus finally broke through the adhesions, and produced diffuse peritonitis.

Moreover, the bacterial finding of streptococci and coli bacilli in the perityphlitic abscess is typical, and the limitation of the diffuse peritonitis to areas below the omentum is also instructive. This simultaneously prevented the invasion of organisms producing inflammation into the serous surfaces above.

This strong man, aged 31, had previously regarded himself as perfectly well. Nothing indicated the danger in which he found himself and which had existed since the appearance of the fecal calculus, the tim when this had formed being impossible to determine. The disease appeared acutely with fulminant symptoms.

Severe abdominal pain with tense abdominal walls, fever and vomiting form the characteristic triad in the first phase of the disease; less rapidly does meteorism appear. This depends upon the fact whether the inflammation of the serosa quickly spreads or remains local. Peritoneal meteorism is peculiar. The abdomen is uniformly distended, balloon-like; the muscles as well as the rest of the abdominal walls are tense. It must be added, however, that in spite of the excruciating pain upon touch there is no sign of contraction of the abdominal muscles, of the "muscular resistance" (*défense musculaire*) which is so common on pressure in other forms of abdominal pain, particularly when circumscribed. The same is true of the diaphragm; it is forced upward, the muscles are therefore elongated and tense; but there is no evidence of active contractions. Abdominal respiration ceases; gradually then, as may be recognized by the limits of percussion, increasing loss of muscle tonus is added. In this case the autopsy showed that the peritonitis had not advanced up to the serosa of the diaphragm.

The excessive abdominal pain, increased by movement and on the slightest pressure, caused the patient to remain motionless upon his back and to avoid the slightest movement of the abdomen either by speaking or coughing.

At the start the temperature was uniformly high, but later remissions in the pus fever were recognized. The pulse from the onset was comparatively frequent, regular, and somewhat tense.

The vomitus was at first composed of the gastric contents, then bile of a peculiarly pure, grass-green, biliverdin color mixed with a yellowish chyme-like material, and in the later stages of the disease showed thin masses having a fecal odor (ileus paralyticus). In regard to the dejecta, the two passages at the onset of the disease pointed to increased peristalsis; this was of short duration, soon changing to the opposite condition, and until the rupture of the perityphlitic abscess absolute constipation existed.

Among these signs pain, either spontaneous or upon touch, a rise in temperature, increased frequency of the pulse and, in general, the signs of severe illness, are to be looked upon as the local and general symptoms of a severe septic inflammation; vomiting, at least in the first stages of peritonitis, was due to decided reflex irritation of the numerous branches of the peritoneal nerves; the fecal discharges at the onset may be explained, but by no means invariably, as due to peristalsis acting reflexly. The constipation which followed this, however, as well as the meteorism, must be attributed to a hypotonia and paralysis of the musculature of the intestine by collateral edema.

Pain upon urination and strangury were due to inflammation of the peritoneal coat of the bladder, in which a noticeable irritation was produced by slight distention as well as by contraction of the bladder. The albuminuria was the well known infectio-toxic "febrile" form; indicanuria was in proportion to the fecal stasis.

In the course of the next few days a new symptom was added to this group: Exudation, which was demonstrable both by palpation and percussion. It was the natural consequence of inflammation of the peritoneum, and was both of diagnostic value as indicating general peritonitis and of special value in that, more definitely than the pain, it pointed to the original seat of the affection, which, according to present indications, could only have been an internal incarceration following right-sided inguinal hernia, or femoral hernia, or appendicitis. As neither the history nor the general status (normal condition of the hernial rings) furnished any points of support for the first view, only the diagnosis of appendicitis, that is, of perforation of the appendix, could be made with that degree of certainty attainable in diseases of the abdominal cavity in general.

After the appearance of these symptoms, a more or less firmly adherent but limited perityphlitic abscess, and a less intense although well developed peritonitis in this region, were assumed; the latter, notwithstanding the general painful meteorism, was not necessarily diffuse in the strict sense of the term; the omentum often protects the upper abdominal cavity from infection, as was proven in this case at the autopsy. It is possible that this diffuse peritonitis, which did not in the early period of the affection extend beyond the limited local focus, was not due to the intestinal contents and to bacteria, but chiefly to bacterial toxins which arose from the circumscribed original focus. This fact is pointed out by the prompt retrogression of the diffuse peritoneal symptoms after rupture of the abscess; the diffuse peritonitis of this first stage might then be designated a non-bacterial "*chemical*" inflammation, according to the terminology now in vogue; finally, it was positively a bacterial infection, although the post mortem finding of bacteria in the distant folds of the peritoneum is not proof of this; we know that during the terminal agony or after death these may wander a long distance from the perityphlitic focus.

After the symptoms of local and general inflammation with their secondary signs in the stomach and intestine had lasted for six days, suddenly a complete change took place: The nervous, anxious, extremely distressed patient became feeble and scarcely complained at all; his formerly congested face was pale and elongated, the nose pointed and cool; the skin lost its turgescence and warmth, and was covered with a cold sweat; the bodily temperature also fell, the pulse became small and frequent but remained quite regular, the abdomen became softer and to a great extent lost its sensitiveness; the vomiting decreased to a few painless attacks, and singultus disappeared: A picture which, to a certain extent, is a combination of collapse and narcosis although not to the degree of profound loss of consciousness, being the picture of an intoxication in sharp contrast to the preceding febrile state. Just as the affection had suddenly developed to its full height at the onset of the disease, and much more swiftly than, for example, is the case in phlegmon of the external walls, so with extraordinary rapidity did the clinical picture assume a new type. In this respect we must consider the very great area of the peritoneal folds, their numerous lymphstomata, and their intimate relation to the circulation, and we are impressed with the fact that fluids and soluble, as well as formed, products are rapidly absorbed by the peritoneum.

Somewhat less rapidly than this, but nevertheless in the course of a few hours, another change took place, a favorable turn following the rupture of pus into the intestine. Here we were dealing with a well known and familiar phenomenon; if this occurs in the peritoneum the effects are particularly well marked; similarly as in the case of a phlegmon which rapidly disappears with the discharge of pus even although the inflammation extend beyond the pus focus, the symptoms of diffuse peritonitis promptly disappeared after the rupture. Very likely, as has already been stated, the symptoms of diffuse peritonitis in the first stages of the disease are to be referred to a chemical inflammation of the serosa, i. e., one due to toxins and without the ingress of bacteria; and it must be remembered that the clinical picture of this chemical peritonitis cannot be differentiated from that of the severe bacterial form. With the rupture of the abscess, the entrance of poisons into the free peritoneal cavity, and their resorption by the extensive peritoneal surfaces, as well as the vomiting and the intestinal paralysis, ceased. The taking of nourishment again became possible.

The point of rupture formed adhesions, the natural drainage of the peritoneal ichorous focus ceased, perhaps a new influx of inflammatory material from the perforated appendix also took place. There was a fresh relapse of the local peritonitis which extended beyond the boundaries of the limiting adhesions, and permitted the invasion by bacteria of the free abdominal cavity. This time the severe toxic picture of collapse immediately followed, and with marked decrease in cardiac strength led to death.

Doubtless the patient might have been saved in the first stages of the disease by the evacuation of the abscess; the incision would at first have acted similarly to spontaneous rupture into the intestine, but the relapse would have been prevented by permanent drainage, and a radical cure might have been brought about by the immediate or subsequent removal of the appendix.

Opium, no doubt, had a favorable effect upon the affection. By relieving intestinal irritability, and by bringing about a mild degree of narcosis, the patient was kept quiet and this materially assisted in limiting the severe perityphlitic suppuration in the first stage of the disease. If, as it unfortunately happened, the point of rupture had not immediately closed again, if it had remained open until suppuration ceased and contraction and healing of the perforated appendix had taken place, opium would have been regarded as instrumental in saving the patient, and unquestionably, at least to some extent, justly so. Among other factors in the treatment, the relief to the intestine by the suspension of nourishment was of paramount importance. The subcutaneous saline infusions had an obvious but, naturally, only a transitory effect.

I have described this case in detail because it is calculated to exhibit to a great extent the present status, and our views, of the processes in acute peritonitis. The case occupies a middle ground between the very acute and the extremely gradual cases of perforating peritoneal inflammation; according to the valuable classification of Mikulicz it should be placed midway between an acute diffuse purulent and a progressive purulent peritonitis. The infection of the peritoneum at the onset was gradual, and not due to a gross perforation, for it may be assumed that the adhesions which paved the way for the favorable process in the severely infected region formed from the beginning, probably before the actual appearance of the affection, and therefore were unnoticed even by the patient himself.

The rapidity with which the peritoneum is invaded by the chemical bacterial infection and the general toxic action of the masses dominate to a preponderant degree the course of perforating peritonitis. In individual cases the virulence of the bacteria is naturally very different, and without doubt it has the greatest influence upon the intensity and extent of the inflammation, upon the quantity of toxins absorbed, and therefore upon the course of the affection; but in many cases it is probably only a secondary factor, for we know that the virulence of bacteria to a great degree depends upon the conditions under which they develop.

If, following this case which occupies a middle position, we consider some others which in an unfavorable as well as favorable sense more properly belong at the end of a series, the importance of this fact, that infectious processes in the peritoneum rapidly implicate its surroundings, will become still more evident.

I shall first quote a very characteristic case of Mikulicz's.[1]

CASE II.—A man, between 25 and 30 years of age, was brought to the hospital in profound collapse; his speech was an idiom very difficult to comprehend, and only after great effort was it learned that for seven years he had suffered from an affection of the stomach, but had suddenly become very ill only a few hours before.

His face was sunken, the extremities were cool, the pulse was 120 and moderately full. He repeatedly vomited masses having the odor of wine. The abdomen was uniformly distended and tense, painful upon palpation, with distinct signs of tympanites. A trial puncture by means of a fine cannula permitted the escape from the abdominal cavity of gas which was inflammable and had the odor of alcohol; after the puncture subcutaneous emphysema occurred in the surroundings of the opening.

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[1] *Volkmann's Vortr.* 1885, Nr. 262.

According to all the signs, a diagnosis of perforating peritonitis was hardly questionable, but the origin was not clear.

The same evening laparotomy was performed. Incision was made in the median line from the navel to the symphysis; upon opening the peritoneum gas and a great quantity of fluid of a coffee-brown color and with the odor of wine, also undigested rice granules, escaped. The entire abdominal cavity was filled with this fluid; the intestine, except for isolated superficial ecchymoses, was of normal appearance. Fluid oozed from the gastric region, and, after enlarging the incision, a slit-like opening 6 to 8 cm. in extent was found in the lesser curvature of the enormously dilated stomach near the cardia.

The stomach was emptied, the opening closed, the abdominal cavity washed with thymol, and the intestines cleansed.

Collapse followed the operation and, in spite of stimulation, death occurred three hours later.

At the *autopsy* a greatly distended and flabby stomach was found, with normal mucous membrane borders around the previously mentioned slit-like opening which had been sewed up; no ulcer; in the vicinity of the posterior upper angle of the wound a striated cicatrix about the size of a ten cent piece, and having no connection with the wound.

The peritoneum everywhere was smooth.

Here, in the course of a few hours, the *severe picture of collapse* developed, resembling that of our first case, with positive local signs of diffuse peritonitis. The operation, which was certainly indicated, may have slightly hastened the end, but, even without this, life would have terminated in the course of a few hours. Rupture of a dilated stomach in the neighborhood of an old cicatrix, undoubtedly due to a former ulcer, was found. There was no gradual inflammation continuing from the serosa, but, on the contrary, a sudden rupture into a previously perfectly normal peritoneum, perhaps due to the cicatrix of an ulcer, with immediate and overwhelming flooding of the peritoneal cavity with the gastric contents.

In rapid sequence was a painful distention of the abdomen with accompanying fulminant symptoms which clinically resembled peritonitis and which, if life had continued, might without doubt have been considered the precursors of this affection, also an immediate and very grave general condition which the earlier physicians designated as "reflex" or "nervous" shock, and the newer school is inclined to attribute to a "septic" intoxication; to this a third fundamental condition was probably added: Congestion of the vessels in the splanchnic area in consequence of congestive hyperemia of this vascular region.

The designation, "perforative peritonitis," is naturally unsuited to a case of this kind, for the patient died before the appearance of inflammation, consequently, not as its result; the most recent designation, "peritoneal sepsis," according to what has been above stated, is, in our opinion, too one-sided. We are evidently dealing with a most unusual case of characteristic of puerperal peritonitis to which various authors, and recently Nothnagel, have called attention.

At the time in which this case occurred, and for such cases, surgical treatment was seldom resorted to; in the case in question it could have been of little avail. Internal treatment was entirely without effect.

These peritoneal inflammations which start from the point of invasion and rapidly implicate large areas of the peritoneum are in contrast to other forms in which there is a more or less conspicuous tendency to limitation. I shall first quote a classical case reported by Mikulicz.[1]

CASE V.—A school-boy, aged 15, was attacked without prodromes by severe abdominal pain and vomiting; in the next few days the abdomen became distended and there was moderate fever. A fecal evacuation occurred daily.

Upon the fifth day: Pulse 100, quite strong, temperature somewhat elevated. Abdomen uniformly distended and everywhere sensitive to pressure, particularly in the ileo-cecal region, where there was increased resistance and limited dulness extending from two to four fingerbreadths from the crest of the ilium and reaching up to the lumbar region.

Incision in this region was followed by the discharge of 200 c.c. of thin fluid pus from a cavity limited by loosely adherent intestinal coils and omentum. The cavity was packed with iodoform gauze and opium and moist heat were ordered.

No noteworthy improvement followed this, but the dulness increased; there was bilious vomiting, no flatus. For this reason upon the following day, the seventh day of the disease, the first incision was extended downward and toward the median line. Ichorous pus exuded from every area limited by fibrino-purulent adhesions of the appendix, several coils of the small intestine and the omentum; the appendix, which was 8 cm. in length, showed a perforation at its middle and adjacent to this a fecal calculus. The operation consisted of resection of the appendix, intestinal suture, careful cleansing (boric acid solution), and packing with iodoform gauze.

The meteorism and the sensitiveness slowly decreased; flatus and later a fecal movement were discharged, but vomiting still persisted. The temperature remained at 100.4°; the pulse was somewhat lower, 104 to 116.

This improvement continued for six days and up to the twelfth day of the disease, when, with a slight rise in temperature and increase in pulse rate, a new area of dulness with resistance and pain upon pressure appeared over Poupart's ligament on the left.

Upon the fifteenth day of the disease another incision was made: A half liter of foul-smelling pus was evacuated from a cavity between adherent intestinal coils which extended into the false pelvis. The cavity was irrigated and drained, and improvement followed.

Upon the nineteenth and also upon the thirty-third day of the disease, accompanied by mild febrile symptoms, two new exudates appeared, the first anteriorly in the median line between the navel and the symphysis, the second upon the right in the true pelvis, and this could be reached from the opening of the second incision. Both were incised and discharged pus. Drainage.

Later still there was a small pleural exudate upon the right side which healed spontaneously, the patient leaving his bed after three months; there was still a fine intestinal fistula originating from the stump of the appendix; it soon closed. A few attacks of colic occurred, then convalescence and restoration to health followed.

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[1] *Mikulicz*, "Ueber die operative Behandlung der Perforationsperitonitis." *Verh. der deutschen Gesellschaft für Chirurgie*, 1889.

This is the case which, if I am not mistaken, Mikulicz denominated as a so-called *progressive purulent form of perforating peritonitis.* Here we may assume that, prior to the manifestations of the affection, toxins or isolated bacteria entered the wall of the already changed appendix and produced a mild plastic peritonitis encircling it. The rupture of the appendix, with which the obvious symptoms appeared, occurred in a region preformed by adhesions; the adhesions, however, were not close enough for chemical poisons, perhaps were not even sufficiently invulnerable to microbes; this is proven by the immediate symptoms of general peritonitis; we have here the typical picture of attenuated general peritonitis (either chemical or bacterial) in the area surrounding a focus of intense inflammation not sufficiently limited by adhesions. We may consider that this diffuse attenuated inflammation was due to toxins which constantly traversed the actual focus composed of pseudo-membranes, and that later bacteria also now and then penetrated this wall and produced the various fresh suppurations some of which extended to distant regions of the abdominal cavity. It is also possible that bacteria were present from the onset, and gradually, in isolated areas, found especially favorable soil for their growth. At all events there was but a moderate local and general toxic effect, and this is obvious from the fact that with slight interruptions feces and flatus were discharged, that the fever was not high, and that not the slightest sign of peritoneal sepsis was ever observed.

The peculiar features of plastic inflammation, which in itself produces resistance, but which may, under some circumstances, be again overcome by the toxins of the disease, we shall meet again in a more distinct form and running a much slower course in the description of tubercular peritonitis. In this case everything occurred in very rapid tempo. Anatomically it is especially interesting to note the important rôle which the omentum plays in limiting inflammations in the abdomen.

As an aid to the diagnosis continuous, persistent, deep palpation (also per rectum!) is very valuable, as is also deep percussion of the entire abdomen in the prompt recognition of pus foci. The focal treatment of the affection is very interesting; the radical procedure of loosening the adhesions at the onset, with the toilet of the entire abdominal cavity, could hardly in this case have produced such brilliant results.

This case, in fact, proved to be conspicuously "progressive." This progression may also take place after apparent consolidation of a local perityphlitis, and with such rapidity that a sudden rupture of the pus focus is simulated.

An observation of Sonnenburg's is a characteristic illustration of such a course:

Case VI.—An apprentice, aged 16, previously well, was attacked with severe pain and vomiting. Upon the third day he was quite ill. The temperature was 101.7°, there was congestion with increased respiration, and a full pulse of 100.

Upon the fourth day an area of resistance about the size of a plate with dulness below the right arch of the ribs amounting to two fingerbreadths and moderately sensitive to pressure was observed; the rest of the abdomen was soft and only slightly sensitive; examination per rectum negative; urine only upon catheterization. The temperature had fallen to 100.8° and, after a careful emptying of the rectum by means of a glycerin suppository, gradually fell to 99.3°; simultaneously the tenderness of the abdomen entirely disappeared but the resistance and dulness upon the right side remained unchanged; the pulse was normal, the appetite better, the subjective condition excellent. Upon the following night and the evening of the sixth day of the disease the picture of severe diffuse acute peritonitis suddenly developed with rapidly rising temperature, a pulse of 140, and great restlessness; the next morning the patient was in collapse, the entire abdomen was sensitive, dulness was distributed; at midday the temperature and pulse fell, the abdomen became softer; during the night the condition became worse and death occurred early upon the eighth day of the disease.

Sonnenburg assumes in this patient, who declined operation, the rupture of a perityphlitic abscess into the free abdominal cavity, and he is probably correct.

The patient was not operated upon in the first stage of the disease, the time chosen in by far the majority of all cases of perforating peritonitis which from the onset show a definite localization, in which the rest of the abdomen is not implicated, and in which the general symptoms steadily improve. A further limitation and a transition into a relatively non-irritating chronic stage or even a cure was expected. That, however, we cannot count upon this with certainty is demonstrated by this case; and it proves how difficult it is to prognosticate the course of such an affection. Such cases are calculated decidedly to bias the opinion of those who see them as to the advisability of operative interference.

Fortunately, however, such cases are infrequent.

In comparison I shall relate the brief clinical history of a case of moderate circumscribed perityphlitis, and one that remained such. This closes the series of the forms of peritonitis arising from inflamed neighboring organs, the series beginning with the severest cases and ranging to the mildest.

Case VII.—H. G., a robust student, aged 22, in the last five years suffered three times from "appendicular inflammation" of brief duration, the last time, according to report, accompanied by a chill and high fever. He was seized in the evening after a fatiguing bicycle ride with a chill, followed by fever, repeated vomiting, and severe pain in the right side of the abdomen; the next morning the temperature was 103.2°, the pulse 120, the general condition moderately severe, violent pain; he vomited bile, there was no fecal movement.

The tongue was thickly coated; between the navel and the spine of the ilium an enormously sensitive area of resistance about the size of a small apple was found. The surrounding parts were also sensitive to pressure as well as the posterior lumbar region. The hernial rings were free; no bladder symptoms. The rest of the abdomen was soft and not sensitive to pressure. The treatment was by ice, small doses of opium; no food allowed.

Upon the second and third days of the disease, while the vomiting had ceased, the temperature remained between 100.4° and 102.2°, the tumor had enlarged to the size of a small plate, was flat, coarse, very sensitive, immovable, with a dull note upon percussion; no fluctuation. No fecal movement, great thirst. The rest of the abdomen was not involved. The patient was quite ill and in severe pain. Moderate amounts of opium were ordered.

Early upon the fourth day, the temperature was 100.2°, in the evening 101.1°; there was no further enlargement. In the evening a compact, dark-brown stool was voided, and in the night another, of pappy consistence, which was not seen. The pain was slight; the temperature had fallen to normal; a general improvement in the condition was noted.

From this time there was no fever, the bowels moved daily without recognizable pus or blood, and the tumor steadily decreased in size; hunger reappeared, and within eight days, except for a deep-seated indistinct resistance over McBurney's point, all the symptoms had yielded. This resistance was demonstrable for some time; then it, too, disappeared.

It had been my intention to propose an operation after the attack abated, but I relinquished this plan in consequence of the complete disappearance of the symptoms.

From this time the patient continued perfectly well for six years.

This case belongs to the extremely full category of moderately severe cases of limited perityphlitis. Perforation is here most unlikely. The condition is probably due to chronic, paroxysmally exacerbating attacks of appendicitis without fecal calculus, but perhaps a slight stricture. In the last attack (whether in the former is questionable) a purulent periappendicitis surrounded the isolated appendix to which adhesions had long since formed, and, according to the position of the tumor, probably also the medial side of the cecum. The quite fulminant discharge of pus in the following days did not rupture the barrier, but only strengthened it. There was no fluctuation. The change in the condition was probably due to a rupture into the colon which rapidly drained the abscess, and in consequence healing soon took place. Subsequently the fresh inflammatory formation was absorbed, and the appendix—as for six years nothing abnormal could be felt, there was no sensitiveness, and no relapse occurred—was probably obliterated.

A favorable course was looked for, although it could not be *prognosticated with certainty* because two milder attacks had preceded this, the disease setting in with moderately fulminant symptoms, without implication of the rest of the abdomen, and without any signs of peritoneal collapse, and because a tumor at once appeared which was very coarse and slowly increased in size. The probability of a favorable course became still greater upon the fourth day when it was observed that the tumor no longer increased in size and the fever declined; but even then the prognosis could not be regarded as quite certain, and the disappearance of the fever and the return of a feeling of well-being did not remove our anxious fear of a relapse, possibly of a still more serious character. This, however, was, according to general clinical experience, to a high degree unlikely.

I am strongly tempted to present as complete a picture as possible of the numerous varieties of peritonitis by quoting still other pathologic pictures. This temptation must be resisted for it would lead us too far from our subject; to be exhaustive in any sense it would be necessary to cite so many isolated cases that the result would be confusion instead of clearness.

Neither is it my object to present a description uniformly elaborated in all directions like a text-book description. On the contrary, when referring to previous histories, points will be described which are regarded as essential to the etiology and to the clinical picture, and which will chiefly aid us in deciding upon the treatment that is to-day recognized as fundamental.

In clinical histories we have learned to recognize inflammations of the appendix, gastric and intestinal ulcers, and septic infections of the uterus as causes of peritonitis; these are partly caused by perforations, partly by propagation through the lymph-vessels. We must still mention other frequent sources of peritonitis: The biliary passages whose sterile contents cause little or no chronic inflammatory irritation, but which, when infected and according to the virulence of the infectious agent (mostly bacterium coli) may cause the severest acute peritonitis; the duodenal ulcer and ulcers of the large intestine, traumatic lacerations of the stomach and intestines; also the important strangulations of the intestine particularly herniæ with their sequelæ of permeability, infection, an necrosis of the paralyzed and inflated intestinal coils; ulcerations an pericystitic abscesses of the urinary bladder, purulent perinephritis, pu tubes, abscesses of the spleen, of the liver, of the pancreas, of the lymph glands of the abdominal cavity; the spleen and glands may also come int question, particularly after enteric fever, the former after severe malari In the newborn the navel or the umbilical vein is sometimes the poin of origin for septic infection; in young children ulcerative prolapse the rectum. Finally, we may mention inflammations from the pleura an pericardium by way of the diaphragm; here, particularly in infancy, t pneumococcus frequently plays a rôle.[1]

Peritonitis originating from the vessels has already been mentioned,- that starting from the umbilical vein of the newborn; in a similar mann septic thromboses from branches of the vena cava and from the port vein, usually the direct result of neighboring suppurations, may arise a cause disease of the peritoneum, more rarely septic emboli from the mese teric arteries in general sepsis. Infection of the peritoneum by circulati toxins from the blood without local vascular disease has a special action,- certainly it is extremely rare. That which was formerly confounded wi it must be due to some neighboring focus in the peritoneum that may not even be detected in a careful autopsy; thus "typhoid peritonitis" is explained by the finest intestinal perforation, or the permeation of the serosa of the intestine without perforation, or by an abscess in the lymph-gland, etc.; malarial peritonitis by a focus in the spleen, and the septic form by a small vascular thrombus, etc. Only in acute articular rheumatism does it appear certain that the toxin passes from the blood into the peritoneal cavity as well as into the joints and all the serous cavities.

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[1] *O. Vierordt*, "Die Natur und Behandlung der Pneumokokkenempyem *Deutsch. Arch. f. klin. Med.*, Bd. LXIV.

Chronic affections of the abdominal organs, such as tuberculosis, actinomycosis, gonorrhea, will be discussed later.

A review of this array of dangers which threaten the peritoneum reveals to us the extraordinarily varied nature of the actual originators of inflammation: The chemically high-graded and toxic gastric contents, the intestinal contents with their chemical substances and putrid bacterial admixture, from which, if sufficient time be allowed, pathogenic microörganisms are first cultivated in the inflammatory fluid; the appendix, of which almost the same might be said, but which also in its wall or in its lumen furnishes more or less pure cultures of pus organisms; the many other suppurations in which the attack sometimes appears to result from a gradual change of the bacteria to the highest degrees of virulence. Among the pyogenic organisms: Bacteria coli with their very varying toxicity, streptococci and staphylococci, less frequently pneumococci, very rarely gonococci and typhoid bacilli; furthermore, however, bacteria of decomposition of all kinds, in part the anaërobic.

Various as are the products capable of producing inflammation of the peritoneum, and innumerable as are the roads by which they may reach this tissue, everywhere, however, two circumstances especially influence the severity of the inflammation: The activity of the germ of inflammation and the rapidity with which it reaches the peritoneum. In regard to the latter, except in external trauma, it is nowhere greater than when one of the numerous hollow abdominal organs perforates into an entirely unprepared, that is, normal, peritoneum. Extreme examples of this kind are traumatic lacerations of the stomach and intestines, ulcer of the stomach, the duodenum, or small intestine, soon rupturing into the serosa and extending far down into the depths of the abdomen, a completely gangrenous appendix inflamed by the pressure of a fecal calculus and necrotic, in which rapid thrombosis of the vessels has occurred, or an empyema of the appendix or gall-bladder rupturing from the pressure of the eroding pus. Here usually large amounts of the contents of these organs are suddenly discharged into the free abdominal cavity; they unfold local and general toxic effects, and produce such similar clinical pictures that their differential diagnosis may occasion the greatest perplexity. And yet we are dealing with very different factors which have entered the peritoneum:

in perforation of the stomach, with the chemical constituents of the gastric contents and an admixture of bacteria in which specific pathogenic bacilli have from the onset certainly not been in the majority, and which—since we are almost always dealing with a preceding ulcer of the stomach—have usually lost much of their vitality and virulence under the decided influence of hydrochloric acid; in a rupture of the appendix occurring under the circumstances just mentioned, we are frequently dealing with a "putrid" infection in which besides the pathogenic bacteria the saprophytic also at first play an important rôle, and with substances which produce chemical and mechanical irritation. In other cases, naturally, within the lumen of the appendix or in its wall the bacterium coli or the streptococcus has developed to a high degree of virulence, and a more or less pure bacterium coli or streptococcus peritonitis develops. If, on the other hand, an inflamed purulent gall-bladder ruptures, a pure culture of less virulent coli bacilli enters the peritoneal sac with the pus, but nevertheless this also results in peritonitis.

Since the well known experimental investigations of Wegner, Grawitz and others, we understand that the animal flora of the peritoneum renders inert large numbers of microörganisms, partly by the bactericidal effect of its secretion, partly by the very rapid and intense absorption into the lymph-spaces, especially in the diaphragm. We know also, chiefly from the investigations of Grawitz, that chemical or physical substances which injure the epithelium diminish the protective power of the serosa, and it is this circumstance particularly which permits the development of bacterial peritonitis. These views have been based upon experiments and pathologic conditions both in animals and man. Clinical experience, however, if it be not too strongly influenced by the suggestive effect of these animal experiments, cannot escape the conviction that in human patholog the protective power of the normal peritoneum is not great. Certainly th surgeon, after the necessary toilet of a fibrino-purulent intestinal coil, see the peritonitis disappear. But here we are not exactly dealing with normal peritoneum producing and absorbing serum; but other curativ factors are operative: Adhesions and the action of drainage. On the othe hand, wherever in human pathology bacterial products of inflammatio attack the normal peritoneal surface, there peritonitis generally develo very rapidly.

Valuable experiments by Silberschmidt[1] have shown that the con bination of deleterious agents present in non-sterilized feces may, in fro twelve to eighteen hours, cause the death of the experimental animal purulent peritonitis. Only pathogenic bacteria are capable of produci a rapidly fatal diffuse peritonitis [illegible], provided they do not fi their way by a single injection but, being enclosed in an animal membra

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[1] Silberschmidt, [illegible] 1. Reihe, Heft

(fish bladder) *gradually penetrate to the free abdominal cavity.* Wieland[1] has conducted experiments of this kind; they are of great value in the study of these conditions; for they show that a minute change in the previous experimental arrangement is sufficient seriously to implicate the peritoneum which was at first so resistant, and to destroy the life of the animal; and, above all, they have the merit, as in Silberschmidt's experiments, of closely resembling the conditions in human pathology; for, if highly virulent bacteria gradually approach and penetrate the lymph-spaces of the uterus or the wall of the appendix and enter the peritoneum, we actually have conditions similar to those in Wieland's tests.

In this connection let us glance over the history of Case No. 3, that of puerperal streptococcus peritonitis! It is a typical example of a diffuse fatal peritonitis due to a pure bacterial culture without the aid of "irritating substances"; and if, in accordance with the experiments of Grawitz and Wegner, we accept the theory of the attenuated resistance of the puerperal organism, we cannot readily understand it. Neither is this assumption borne out by the facts when a phlegmonous, erysipelatous inflammation of the wall of the appendix without perforation produces an extensive purulent peritonitis in the normal peritoneum (notwithstanding its undoubted powers of resistance).

The result of all this is that we must not estimate too high the power of the normal peritoneum to resist microbes; i. e., above all, to destroy them by absorption; if this be regrettable, it also has something in its favor. For we know and can prove by the previous histories that a massive absorption of toxins from the peritoneum leads to the severest form of peritoneal sepsis, and this we shall later discuss.

Protection by the normal absorption of the peritoneum, therefore, does not play the important rôle in pathology which was formerly often ascribed to it. *All the more important are the protective functions of the inflamed serosa.*

Before the peritoneum becomes implicated by inflammatory processes in its vicinity, changes are produced which may best be compared with the changes in the skin and subcutaneous cellular tissue surrounding a pus focus. In the peritoneum hyperemia develops, also a fluid exudate generally deficient in cells, which here, however, flows into the free abdominal cavity; this exudate may be entirely devoid of bacteria, in which case to-day we speak of a "chemical peritonitis"; rarely in these cases does the exudate become rich in cells, or even purulent. There is no doubt that these fluid effusions are of some value in rendering inert the phlogogenic microbes which enter the abdominal cavity from the focus of inflammation, and eventually perforate.

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[1] *E. Wieland*, "Experimentelle Untersuchungen über die Entstehung der Peritonitis." *Mitth. a. d. Schweizer med. Instituten*, Bd. II.

But this action, as already stated, must not be estimated too high; all the greater, however, is the protective effect of plastic exudates. Their production is the especial property of the serous membranes; they may develop even a few hours after the beginning of an inflammatory irritation, and may appear as swelling and redness of the skin surrounding a pus focus already formed around an inflammatory focus which threatens to attack the serosa. These plastic exudates, however, confer protection by leading to adhesions, and later, by the extension of these, to adhesions of the serosa with neighboring serous surfaces. Preferably they unite the anterior surface of the stomach with the abdominal wall, the greater curvature with the colon, the gall-bladder with the colon, etc.; they produce membranes which usually extend from the intestine at a point opposite the mesenteric attachment to the abdominal wall; they form a sac around the inflamed appendix which, if ensuing perforation occur, is calculated to absorb the putrid masses and the pus which these develop.

But, rapidly as these structures form, they nevertheless require time to attain the necessary degree of firmness.

Concerning this point, the clinical histories above cited should be investigated, the autopsy findings in the rapidly fatal cases, Nos. 2 and 3, and the findings at the operation and at the autopsy in cases Nos. 1, 4, and 5. The great importance of this circumstance is obvious, and since adhesions which exist before the rupture, if firm, afterward become stronger, sometimes in a very short time, we find perfectly firm encapsulated pus cavities in various regions of the abdomen; these encapsulated pus foci are probably most conspicuous alongside the cecum, behind the stomach as subphrenic abscesses, and in the true pelvis.

It is quite possible that occasionally even in perforation,—for example, of an ulcer of the appendix with a normal peritoneum, therefore, in the free abdominal cavity,—these adhesions may form rapidly enough to encapsulate putrid masses; it must here be taken for granted that these are quiescent, and this above all presupposes absolute quiet on the part of the patient and absence of motion in the intestine. Probably, however, these adhesions are generally preformed before the perforation; naturally, these are facts beyond our knowledge.

The more infectious the pus that has formed, and the greater the pressure under which it develops, the tougher must be the adhesions to protect the free abdominal cavity.

Let us again review cases Nos. 1, 2, and 5; we see that at first typical encapsulation took place; later, however, the adhesions were not compact enough. They either ruptured mechanically by pressure of the pus, by peristalsis or other causes, or were obliterated by the functions of the white cells and bacteria, and thus, finally, they paved the way for the entrance of inflammatory products into the free abdominal cavity, provided that, in the meantime, additional adhesions had not formed. In the former case a general peritonitis secondarily developed, in the latter case there was a relapse of circumscribed peritonitis. In both cases we see the condition which Mikulicz has called the *progressive purulent form* of acute peritonitis.

The occurrence of such secondary ruptures depends, therefore, partly upon the relation between the internal pressure of the abscess and the firmness of its wall, and partly upon vital and chemico-physical processes in the wall itself.

As auxiliary factors of decided importance the intensity of intestinal and gastric peristalsis, possible external shock, etc., injury by purgatives and enemata or even by inunction or massage, must be considered. At all events, it is clear that the firmness of the boundaries is always doubtful, and, especially at the onset of the disease, this can hardly be looked upon as certain, least so with a closed abdomen, and this is of significance in the prognosis of such abdominal abscesses. The clinical history of Case No. 5 is an illustration of the surprises that may be in store for the physician.

This point will be further elucidated under the discussion of the treatment.

Chemical peritonitis has been referred to; this exhibits all the peculiarities of sero-fibrinous peritonitis, it may become rich in cells and even purulent—but bacteria are absent. It is probably brought about by bacterial toxins conveyed from a near focus; chemical peritonitis, therefore, occurs in the surroundings of an insignificant bacterial pus focus of the abdominal cavity; that is, from a periappendicitis, a psoas abscess, an inflamed appendix, an ulcer of the stomach or even an inflamed gallbladder. In the latter case, as we have seen, it is generally plastic. Its appearance in the hernial sac about the incarcerated intestinal coil is characteristic and instructive; here, at all events, the paralyzed intestine is permeable for the toxins of intestinal bacteria and other soluble products, and this is the precursor of permeability for bacteria, and leads to necrosis of the intestinal wall; similar conditions arise in severe traumatic contusion of the intestine.

CHEMICAL PERITONITIS

In *chemical peritonitis* one thing is especially interesting to the clinician: The hyperemia and resulting fluid exudation appear and disappear *according to the focus of origin.* A circumscribed periappendicular abscess may produce a distributed chemical peritonitis in its surroundings; this manifests itself by moderate or even decided sensitiveness upon pressure and distention of the abdomen, and, perhaps, by decreased but, clinically, often threatening symptoms of extensive peritonitis; if the abscess is opened or ruptures spontaneously, these symptoms suddenly disappear. Much of that which the clinician, for want of a better term, has designated as "peritoneal irritation" belongs to the realm of chemical peritonitis. This dependence of chemical peritonitis upon a bacterial focus of origin is due to the fact that the process, being independent of bacteria, does not generate new causes of inflammation, ***is not in itself capable of propagation, or even progressive.*** This does not preclude its occurrence as an accompanying process of great intensity and extension and directly or indirectly extremely dangerous (by moderate meteorism, forcing upward of the diaphragm, etc.). This form of inflammation differs from the metastases containing bacteria of peritoneal inflammation, which may spontaneously disappear provided their action is moderate and they may thus be destroyed by the peritoneum; on the other hand, in many cases they may survive and continue to develop even after the focus of origin has been removed. These points give us important indications for the treatment.

It is true the plastic exudates of chemical peritonitis, as well as those generated by the presence of bacteria, may persist for a long time; they may be organized into pseudo-membranes and indurations long after the source of inflammation has been removed. This newly formed connective tissue, by its bulk, resistance, and indistensibility, even by its cicatricial retraction, may contract, deform, and limit motion, and in various ways may narrow and constrict the abdominal organs and cause adhesions. In the consideration of chronic peritonitis we shall revert to this subject again.

The foregoing description makes clear the meaning of our statement that the severity of peritonitis depends primarily upon two things: Th activity of the septic microörganisms and the rapidity with which the reach the serosa from a focus near the surface of the peritoneum.

In contrast to these numerous methods of infection from neighborin organs are invasions of the peritoneum by means of the blood or lymph channels, which are certainly rare. They occur in acute articular rheu matism and occasionally in sepsis. In the cases in which small encapsu lated empyemata, particularly the metapneumonic, complicate a purulen peritonitis, transmission by means of the blood may also sometimes occu as I have previously stated.[1]

PATHOLOGY AND GENERAL CLINICAL PICTURE

In regard to the pathology and general clinical picture of acute diffu peritonitis, the clinical observations quoted at the beginning of this artic indicate distinctly that two different pathologic conditions are to be co sidered: A congestive inflammatory general condition with specific acco panying symptoms of peritoneal irritation, and the general picture collapse. Mixed forms between these occur, although rarely; as a ru they may be sharply differentiated, but occasionally they rapidly merge into one another.

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[1] *O. Vierordt, Deutsch. Arch. f. klin. Med.,* Bd. LXIV.

The triad of symptoms mentioned in the first clinical history,—severe abdominal pain, fever and vomiting,—has been sufficiently described in the epicrisis of the clinical histories, as well as peritoneal meteorism, which almost always occurs as a fourth symptom. These four symptoms, while not invariable, are found so uniformly in most cases of acute diffuse peritonitis, and they dominate the clinical picture to such an extent, that without a history the status of the patient frequently gives no clue for the differential diagnosis of the origin of the affection. If the history does not aid us, this question under some circumstances cannot be decided; but, as a rule, there are differential diagnostic guides to which we shall later refer.

Regarding the second condition, the seeming collapse, in our clinical histories we note its appearance under very different circumstances: In Case 1 it developed twice from the previously existing congestive inflammation; the first time it disappeared after the salutary rupture of the periappendicular pus into the intestine, and the second time it was the cause of death; in Case 2 (perforation of the stomach) it appeared a few hours after the onset of the affection; here, post mortem, the massive contents of the stomach were found in the abdominal cavity, and the peritoneum was quite smooth and glistening. In puerperal septic peritonitis collapse occurs early, but appears to be modified by the delirium and marked somnolence; post mortem we find a severe, purulent streptococcus peritonitis, but no perforation, no putrid infection.

The symptoms common to these clinical pictures are severe cardiac and vascular weakness, prostration, cold sweat, lowered general and surface temperature, and comparatively mild subjective symptoms; in addition to these a severe, widely distributed peritoneal irritation is common, and also probably the absorption of toxic products.

But, upon closer investigation, these conditions differ from each other, and far more etiologically than clinically. In one case we have the sudden flooding of the peritoneum with the gastric contents: Gastric juice containing hydrochloric acid, albuminates, peptones, salts, etc., and also bacteria which are of diminished vitality, consequently bacterial toxins here play no rôle. In the two other cases we have severe bacterial peritonitis—one being a pure streptococcus peritonitis; in both, especially in the latter, there is profuse absorption of bacterial toxins: Sepsis.

At the present time these conditions, especially in Germany, are regarded from too one-sided a standpoint,—usually as peritoneal sepsis,—and their other aspects are ignored; namely, the effects of shock upon the central organs, that is, the cardiac and vascular centers, originating from decided irritation of the sensory end organs of the sympathetic in the peritoneum, not alone in peracute perforations, but also in peritonitis

DIAGNOSIS

The diagnosis of diffuse peritonitis may be easily made when we consider the totality of the symptoms. In a minority of cases, however, we meet with differentio-diagnostic difficulties which are usually very great.

An acute peritonitis without meteorism, i. e., with a hard retracted abdomen, with excruciating pain in the gastric region, but without vomiting or constipation, and in which there is perhaps a history of ulcer, may simulate a severe cardialgic attack in a patient with gastric ulcer; fever, diffused pain upon pressure in the hypochondriac region, and even in the lumbar region, and the symptoms of collapse will favor peritonitis, but the diagnosis may remain doubtful until meteorism appears. The same conditions are also observed in ulcer of the duodenum.

Severe inflammatory *attacks of gall-stone colic* with a distended abdomen, vomiting, and even fecal vomiting and fever, may cause great perplexity in the differential diagnosis, particularly if the anxious patient states that the whole abdomen is painful upon pressure. The difficulty is, however, at least lessened by the rapid disappearance of meteorism when the attack ceases, and perhaps with the administration of morphin.

In the majority of cases, however, it is difficult to decide whether peritonitis or *intestinal obstruction* is present, particularly if strangulation be suspected, since the latter, as is well known, gradually produces peritonitis. Simple obstruction is readily differentiated from peritonitis by the absence of fever, by the less painful meteorism, by a more or less distinct local peristalsis, by collapse which sets in late or does not appear at all. The obstacles are even greater in the diagnosis of obstruction due to strangulation: Examination of the hernial rings (they must all be examined, and all symptoms of hernia considered) by rectum will sometimes at once decide the question in favor of the latter; but where all these tests are negative or uncertain (as in long-standing, non-incarcerated hernia), in the absence of fixed inflated coils (v. Wahl) or of peristalsis above these (Schlange)—as is unfortunately *frequently* the case—the differential diagnosis at once becomes very obscure; early fever favors peritonitis (initial shock occurs in this condition as well as in strangulation) and hemorrhagic dejecta indicate strangulation, particularly invagination. The longer the condition lasts the more difficult is the diagnostic differentiation, because peritonitis is then added to strangulation. It is obvious that under these circumstances the previous history of the patient is important and occasionally decisive.

The same conditions are met with in the extremely rare cases where an enormous fecal accumulation causes great meteorism, vomiting, sensitiveness of the abdomen, eventually fever, and simulates peritonitis. Uremia also may lead to confusion when accompanied by severe vomiting, meteorism, diarrhea and fever, and when the symptoms of nephritis (acute or chronic) have been insufficiently observed.

PSEUDO-PERITONITIS, PERITONISM

By a pseudo-peritonitis, peritonism (Gubler), we mean conditions in which the combination of abdominal pain, vomiting, a tense and usually distended abdomen, constipation and perhaps even fever depending upon a hysterical basis simulates peritonitis. These cases usually occur in young hysterical persons, not rarely in children; occasionally the causes are mild intestinal and genital affections, retention of urine and feces, slight trauma. The clinical picture may, at the first glance, lead to confusion; but upon careful observation a certain disproportion in the symptoms becomes obvious; for example, tearing pain and continuous vomiting without a sign of collapse; in one of my cases there was severe spontaneous pain as well as tenderness upon pressure with marked meteorism, but, instead of lying still, upon palpation the patient made energetic efforts to rise; this does not occur in acute peritonitis. Other characteristics are the abrupt change in the symptoms, the effect of suggestion particularly on the pain, the absence of cardiac asthenia (although Naunyn saw dyspnea and a poor pulse due to the elevated position of the diaphragm), finally, stigmata and *antecedentia hysterica* must be considered, both of which, naturally, may be absent in the young. Taken all in all, the careful observer is liable to be mistaken the first day of the disease, but, upon the second, one of these signs will lead him to a correct conclusion.

In all of these cases, in which, in contrast to diffuse peritonitis, non-inflammatory or, at least, not diffusely inflammatory affections in the abdomen, or other conditions are to be considered, a rise in the rectal temperature (two or more degrees higher than in the axilla) may be of diagnostic importance, but only when the temperature is very accurately taken by the axilla.

The *diagnosis of the point of origin* is easy, provided the symptoms of the original affection have been previously observed by the physician; otherwise, the diagnosis is generally the more obscure the greater the development of the peritonitis. But even then it may often be made from the history—which, however, may be erroneous (cardialgia and gall-stone colic, ulcer of the stomach and ulcer of the duodenum, an affection of the pancreas, perityphlitis and genital affections, etc.).

It is most important to determine the seat of greatest tenderness, and there eventually to locate an objective point. Some authors have recently attached great significance to the hyperalgic zones of the skin. Examination of the hernial rings and of the vagina should never be omitted; both this and rectal examination are generally necessary for the detection of exudates into the pelvis, and to differentiate perityphlitis from genital and bladder affections.

It must be remembered that the individual abdominal organs, particularly the appendix, have no fixed position; the tip of a long appendix may reach far down into the right iliac fossa, as has been previously remarked. A pericholecystitis will occasionally be situated very near McBurney's point, or may be also somewhat displaced laterally. There is naturally no positive seat for perforation of the small intestine, although typhoid perforation is often found low down upon the left side. Ovarian affections may be widely distributed, particularly toward the ileo-cecal region. The pancreas and duodenum have fixed locations, but here the differentiation from the stomach is difficult.

CIRCUMSCRIBED ACUTE PERITONITIS WITH PARTICULAR REFERENCE TO PERITYPHLITIS

PATHOLOGY

Circumscribed peritonitis is prone to develop when bacteria find ingress to the abdominal cavity and penetrate to regions preformed by adhesions. We have previously seen that in perforation the severity of the infection of the abdominal cavity depends upon the amount of material discharged into it from the stomach and intestines, upon the entrance of pyogenic organisms from the lymph-tracts, upon the virulence of the pathogenic pure cultures which have been previously produced in the tissues. Now, if the perforation be very minute or the virulence but moderate if it take place in an area favorable for deposit (the ileo-cecal region, th pelvis, the subphrenic space), on the cessation, spontaneous or induced of intestinal peristalsis and when the patient remains very quiet—it cer tainly happens that a previously normal peritoneum without adhesion will produce direct epithelial or fibrinous adhesions of its walls, and, i the meantime, will absorb a certain amount of the poison and in thi manner localize the process. The individual parts of the abdominal cavit joined to the mesentery form a fan-like structure which aids us her The clinical picture depends upon how rapidly encapsulation takes plac whether it be complete or partial, whether the toxins do not produ (although their agents themselves do not) a diffuse chemical peritonit beyond the protective wall, and upon the density of the adhesions, tl firmness of their hold, and their further course. Naturally it also depen upon the reaction of the entire organism to the irritation of the per toneum and to the intoxication.

From the different forms of circumscribed peritonitis I shall sing out perityphlitis, and describe this affection in detail. This is the disea which, in the majority of cases, presents itself to the family physicia and it has become a mooted question whether it does not, from the ve onset, belong to the realm of surgery. Besides, it shows the greatest variations, and therefore best illustrates all of the conditions which play a part in circumscribed peritonitis.

We have seen from the history of these cases that circumscribed perityphlitis may be absent clinically and anatomically (Case 3) in the severest, i. e., in the most acute cases of perforation of the appendix; that it may sooner or later develop in diffuse perforation of the appendix (Case 1), and may even appear simultaneously with this or may precede it; that an accompanying general peritoneal irritation may dominate the clinical picture; that, finally, and fortunately in the majority of cases, it may occur alone and exclusively in an otherwise normal belly. In its *course* this focal affection may extend to the surrounding portions of the abdominal cavity. It has just been mentioned that perityphlitis may appear gradually as the actual nucleus of a distributed peritonitis, and when the peritonitis disappears may dominate the picture; inversely, from the originally circumscribed affection one or more serious relapses may unexpectedly occur, perhaps with fatal result.

Which of these extraordinarily different clinical pictures will develop depends chiefly upon the nature of the appendicitis which has produced the peritoneal inflammation (skolikoiditis is the designation proposed by Nothnagel); this collective name, however, includes many varieties. If we leave out of consideration the tubercular, the typhoid, and the actinomycotic affections of the appendix, most of the non-specific cases may be referred to two large groups of causal changes in the appendix: To *fecal impaction*, and to certain *chronic inflammatory* and also *acute "relapses"* of the wall of the appendix.

Proceeding from the most dangerous to the mildest diseases of the appendix, we first distinguish an acute distributed gangrene of the organ with immediate perforation and putrid flooding of the surrounding area; next, the milder cases, i. e., those with gradual onset and, at least in the beginning, less extensive putrid infection of the peritoneum by the perforating, gangrenous or non-gangrenous ulcer. These lesions are almost always due to the greatly dreaded fecal concrement, although it may certainly be absent. We recognize also a gangrenous inflammation of the appendix without fecal calculus, particularly in stenosis, followed by the rupture (which may be gradual) of an empyema of an appendix which is closed at the cecal end by stricture, but also by empyemata and suppurations of the wall of an appendix which, without a gross rupture, permits the passage of pus cocci; nearly all of these cases are due to collections of bacteria (Tavel and Lanz), among which pathogenic microbes that have previously attained decided virulence are found to predominate (almost invariably bacterium coli). But simple phlegmonous or erysipelatous inflammations of the mucosa of the appendix may, without themselves being purulent, produce a suppurative perityphlitis usually charac-

[illegible] with slight toxic action and [illegible]

Passing from [illegible] to the milder forms, we note that the appendix is frequently [illegible] catarrh of the mucous membrane and its numerous glands and [illegible] of the vermiform appendix, [illegible] but also of deeper chronic inflammation of its connective [illegible] (appendicitis granulosa, Riedel). These changes may be the starting point of the above mentioned severe forms of inflammation, either directly or by causing secondary changes: Stenosis and stricture by loss of epithelium, the formation of cicatrix, adhesion of the surfaces of mucous membranes lying opposite each other.

A factor of great significance in the development of these forms of appendicitis is that the serosa of the appendix, and also its mesenteriolum often early implicate the wall of the organ, even by mild chronic inflammatory changes. In these instances the serosa, as we learn from earl surgical operations, is rarely quite normal, but is usually reddened, per meated by numerous vessels, more or less thickened, often unevenly, b the formation of constrictions of the appendix; frequently the mese teriolum is also implicated, being shortened, thickened, bent and tor causing immovability of the contractile organ. Often the entire ma early becomes embedded in new connective tissue, or the appendix its end or at some other part of its length adheres to the cecum, the ili fossa, the ovary or the bladder.

There can be no doubt that these inflammations of the mucous me brane and the early changes in the serosa indicate severe disease. If remember that the appendix is comparatively long and thin, a holl secreting organ, it is not difficult to realize that even a moderate dimi tion of the elasticity of its enclosing serosa, a slight induration of same, but still more an external stenosis and kinking, may very read check the flow of its secretion, as well as lead to circulatory disturban in its walls. Moderate stricture may act like a narrowed foramen u catarrh of the frontal bone; desquamation and serous transudation of tissue may also cause venous stasis; the finding of a penis-like thicken of the process, but even more the microscopic findings of Riedel (rou cell accumulation, disappearance of the glands and *hemorrhages*) n increase this presumption. But these processes take place in the ne borhood of products which mechanically and toxically injure them, in the presence of putrid collections of bacteria; doubtless in the la case the pathogenic bacteria which are always present frequently gain upper hand. We must, therefore, on the one hand, attribute to the [illegible] of the secretion, and on the other hand, to stasis combined the above circumstances, a part in the genesis of all mild and severe fo of periappendicitis which develop without fecal calculi; but also severer forms [illegible] the remarkable finding of acute gangrene without calculus may be plausibly explained by a free desquamation of the organ with limited distensibility, by immediate severe stasis in the region of the fine venous branches of the interior, and by gangrene from the influence of bacteria constantly ready to develop.

The most vital point here is the intimate relation of comparatively mild changes in the appendix which cause no apprehension to the most severe and most dangerous. This is probably due to the fecal calculus which may remain a varying length of time in the appendix without producing symptoms (in some cases large fecal calculi have been discovered accidentally without resultant effect), and then, as a rule suddenly, exhibits decided activity. On the other hand, it may be due to the previously mentioned chronic inflammatory conditions. In regard to the latter we are indebted to Riedel for having systematically paved the way to the recognition of their relations to perityphlitis. He has shown that chronic appendicitis is in some cases a comparatively mild, in others an exceedingly grave, affection. He relates his observations and results of investigation so clearly that I cannot refrain from quoting his remarks on the point in question: "In one case the disease (appendicitis granulosa) leads insidiously and painlessly in the course of years to obliteration; in another, one or several inflammatory relapses occur and obliteration results; in a third the appendix, notwithstanding several relapses, remains in the state of appendicitis granulosa; in a fourth, the lymph-glands become infected; in a fifth, a periappendicular abscess forms which may later be absorbed without a trace of pus appearing in the appendix; finally, in a sixth, the acute inflammatory paroxysm begins with suppuration, even with gangrene; here appendicitis granulosa forms the basis for the acute suppurative attack, usually without fecal calculus, so that frequently the pus is but slightly or not at all putrid; with gangrene that sets in at once it is usually putrid, but not invariably."

If we consider what has long been known of fecal calculi, and what has become obvious from the conditions just described, we come to the conclusion that in by far the majority of cases of severe appendicular peritonitis dangerous to life the disease has existed for a longer or shorter time. For the clinician, however, the important questions at once arise: Can these preceding changes be clinically recognized, or can we, at least, approach more nearly to their recognition? These questions we will discuss later.

If suppuration occurs in any of the previously mentioned regions outside of the appendix, the development and course depend upon various circumstances. The most malignant forms are the putrid infections and those from highly virulent and more or less pure pyogenic agents (bacteria coli, streptococci and staphylococci), the former occurring in gangrene with or without fecal calculus, but usually without, the latter less frequently in a few cases of empyema and phlegmonous appendicitis.

An important and usually decisive factor is the eventual presence and the strength of the adhesions; coarse ones resist the mechanical and vital influence of pus, weaker ones succumb. As auxiliary conditions we must mention the position of the abscesses as related to the cecum: Those medially situated soon penetrate into the free abdominal cavity, those laterally situated toward the sacral hollow of the pelvis are most readily limited. Suppuration between the layers of the mesenteriolum appendicis frequently extends to the retrocecal space and occasionally along the colon as far as the descending colon; thus it may give rise to paranephritis either dextral or sinistral, subphrenic abscess, empyema upon the right or left side; and these may apparently be produced paroxysmally. Suppuration low down in the median line readily finds its way to the pelvis. It is obvious that when pus is found in this locality the original position of the appendix has a decided influence.

Periappendicular suppuration, even when not very extensive, produces desquamation and serous transudation of the surroundings: of the serosa of the cecum and appendix, the preformed adhesions and, possibly, the adherent parietal peritoneum in this region, perhaps also of some of the mesenteric glands; in some cases the connective tissue of the retroceca space becomes involved; all this occurs to a varying extent, and produce the *perityphlitic tumor* in the interior of which suppuration is foun which resembles the purulent nucleus of a furuncle (Sahli). If the pr becomes more profuse the tumor may show fluctuation; it may becom enlarged by an accumulation of feces in the paralyzed cecum, by refl action of the inflammation or by the intestinal paralysis which may very extensive, and by vomiting. Occasionally it may produce dista effects: Engorgement and, not rarely, serous exudation ("chemical per tonitis") of the free abdominal cavity which is usually the result a general toxic action, namely, fever.

As we have just seen, non-purulent inflammatory processes in t appendix cause external suppuration, mild irritative conditions in t vermiform process cause thickening of the serosa and adhesions. Th mild irritations, especially the relapses of appendicitis granulosa, are pr ably capable of producing relapsing, that is paroxysmal, serous tran dation and serous or sero-fibrinous exudates (Sonnenburg) in the s roundings without the formation of pus, and these are clinically obser as tumors; they are characterized by their brief duration, their ra disappearance, sometimes by their sequelæ of marked thickening adhesions.

The variety of conditions produced by circumscribed perityphlitic s puration is partly illustrated by clinical histories; left to themselves, a more or less time they are prone to rupture, generally into the intest especially into the colon, and then heal; they may also rupture ex nally,—through the skin, into the bladder, into the rectum, into a ple cavity and perhaps into the lung; or they penetrate to a greater or less extent within the free abdominal cavity and immediately cause an acute diffuse peritonitis, or, in case adhesions were preformed, a progressive purulent peritonitis (compare clinical history No. 4); in the series of relapses they may produce here and there a number of new foci which result in a more or less rapid destruction of the organism. But metastatic suppurations may also, particularly by rupture through the intestine, force their way outward, and surprising recovery has been observed even in the severest cases; in the first stages of progressive peritonitis, however, we often note a sudden halt in the process, it ceases everywhere except in the perityphlitic center, and even here it finally runs a favorable course or becomes chronic.

Besides this unexpected change for the better there may be an unlooked-for change for the worse; a perityphlitic abscess which has previously been circumscribed may, at any time, after days or weeks, rupture; general peritonitis, sepsis, and death follow. Doubtless this occurrence is most frequent in recent encapsulation, i. e., in perityphlitic abscesses which have existed only three to five days. The longer this period and the firmer the encapsulation, the more unlikely is a fatal rupture; but it can never be excluded with *certainty* while pus is present in the abdominal cavity. It is in putrid suppuration that this condition is always most to be feared.

There can be no doubt that on partial discharge of the pus, for example, after surgical operation, the remaining pus is absorbed, even if the wound be imperfectly drained. Consequent upon operation special conditions are present; congestive hyperemia probably acts by vitalizing all important processes, the absorbent as well as the bactericidal. Therefore in the general application of these observations, particularly when we have the condition of a closed belly, we must be cautious; prior to operation, such a condition is extremely difficult to distinguish. What appears to be absorption may very likely be an unperceived rupture into the intestine. Even more than in the normal peritoneum, we must avoid over-estimating the absorptive activity of the peritoneal pseudo-membranes. We have little certain knowledge regarding these; all is conjecture.

Of other complications produced by pus, only septic thromboses of venous trunks in the portal vein system, or of branches of the iliac, or of this vessel itself, are to be mentioned. The latter may lead to septic embolism of the pulmonary artery, and pulmonary abscess (not always fatal). These thromboses usually result in general sepsis and death.

SYMPTOMS

In considering the clinical symptoms of circumscribed perityphlitis, we must again review the clinical histories: Acute illness with severe pain in the ileo-cecal region, vomiting, a varying temperature and usually rapidly rising fever, but rarely fulminant symptoms; frequently, at first, a few thin fecal movements, followed by constipation which depends upon intestinal paralysis, and then the appearance of a painful, more or less immovable tumor in the region of McBurney's point, which at first increases in size, is coarse upon palpation, but rarely fluctuates markedly; the inguinal glands are occasionally swollen; flexing the right leg often produces deep-seated pain in the belly; if this be very severe it favors paratyphlitis. This is the typical picture to which, under some circumstances, the more or less intense symptoms of a diffuse peritoneal inflammation or "peritoneal irritation" are added.

The case is not always so simple; in all similarly located affections the possibility, and even the likelihood, of perityphlitis will come into mind because this disease is so frequent. But the symptoms will often necessitate careful inquiry; under some circumstances, nothing more can be found than an indistinctly limited resistance deep in the belly and covered by an intestine containing gas,—occasionally not even this.[1] To ensure certainty from the onset in the differential diagnosis an examination must always be made of the hernial rings, of the vagina, as well as of the rectum; vaginal as well as rectal examination is usually to be repeated in the further course (to solve the question of possible descent into the pelvis).

Pain, fever and resistance are the principal symptoms.

Pain.—The pain in the majority of cases at once correctly indicates the location; as a rule it is spontaneously severe and upon pressure unbearable; it is situated near McBurney's point or often somewhat below it. At the onset it may be diffuse, or may be complained of in à paradoxical region (upon the right above, upon the left below, etc.); but, among all the acute abdominal affections with pain, it should be remembered that perityphlitis is the most frequent; it should always be borne in mind. The position and the radiation of the pain from the median line downward, and often associated with dysuria (indicating inflammation toward the pelvis), to the lateral and posterior lumbar region (inflammation in the retrocecal and retrocolic space) are only briefly mentioned. Psoas pain upon flexure of the right side certainly occurs in perityphlitis without decided paratyphlitis. Painful flexure contraction of the thigh, however, and the impossibility of extending it, point with great positiveness to the retrocecal connective tissue. Lancinating pain upon pressure points to pus, dull pain does not exclude it. Very severe spontaneous pain and pain upon pressure with only a small tumor and mild general symptoms are not rarely observed in empyema.

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[1] After this article was finished I read an excellent publication by B. v. Beck upon "Weitere Erfahrungen über operative Behandlung der diffusen eitrigen Peritonitis" (*Beiträge zur klinischen Chirurgie*, 1902), from which I see that v. Beck, in the diagnosis of the point of origin of peritonitis, attaches importance to the previously mentioned circumscribed hyperalgic zones of the skin; he looks upon them as a valuable indication. In regard to this symptom, I have as yet no experience.

Fever.—In regard to the fever, I should like to state briefly that in my experience high fever, according to its duration and type, enables us to arrive at positive diagnostic and prognostic conclusions. But the absence of fever, or a fall in the temperature, must be utilized with a certain reserve. A glance at the clinical history shows that high fever, setting in abruptly with chills, with a parallel increase in the pulse rate, here, as well as frequently in pathology, indicates pus; yet it may occur without pus, particularly in the inflammatory relapses of appendicitis granulosa (Riedel); less rarely is vomiting absent in either case. High fever with a very rapid small pulse, excruciating pain, and severe vomiting usually points to perforation. It will be remembered, too, that the symptoms of septic collapse and the general abdominal signs of perforation occur conjointly with immediate diffuse peritonitis or peritoneal sepsis; when the exudate is limited, severe septic collapse is absent and the abdominal symptoms are slight, or, in case they are well developed, they decline in one to three days, usually with a fall in temperature. But cases midway between this latter picture and that of acute septic peritonitis are not rare. The fever course which indicates rapid limitation is, therefore, that which gradually declines for three, five or more days after the initial height. The local signs accompanying this are usually those of concentration upon the perityphlon. A change at this time with rise of fever and still greater distribution of the process is serious, but, fortunately, infrequent. Gradual limitation corresponds to a sub-continuous range with diffuse abdominal symptoms lasting several days, the fever disappearing by lysis when the limitation has been reached; but even complete circumscribed peritonitis sometimes gives rise to high fever continuing for a week or longer. If collapse is not present sudden decline of the fever almost always indicates perforation of the abscess into the intestine, or one of the other directions indicated. The feces and urine should be watched!

A milder type of fever usually accompanies slight perforations of the appendix or purulent periappendicitis without perforation, but also appendicitis simplex with slight, non-purulent implication of the serosa. Empyema of the appendix presents a varying temperature, usually not high fever, occasionally none at all; sometimes, however, chills and great elevation of temperature are observed.

In the later course a sudden, sharp rise without symptoms of sepsis points to an increase of the purulent inflammation; that is, in accordance with the conditions present and the accompanying local symptoms, to progressive purulent peritonitis, to an extension of the process along the colon, or to subphrenic abscess, empyema, etc.; another decided rise with immediate septic symptoms which at first may be only latent, indicates threatening diffuse peritonitis. But even here there are also intermediate forms: For example, rapid and progressive distribution of the disease without septic collapse.

The long-continued, progressive purulent forms naturally exhibit a long-continued febrile state with great variation in temperature, chills, a sub-continuous course during renewed encapsulation, a decline with possible rupture, etc.

I cannot go beyond these general remarks; it is easy to construct medium courses as types; Rotter and others have no doubt constructed these by carefully utilizing their numerous observations. From a didactic standpoint these types, which are exemplified only in a certain number of cases, are always questionable, because the reader who does not bear in mind the multiplicity of exceptions is always inclined to adhere closer to the type than does the author himself.

Tumor.—The tumor varies greatly in size, varies in position according to its mode of attachment to the appendix and its length, varies in consistency according to the manner of its production. Its walls may be perceptible or may be overlapped by intestinal coils, and in the latter case, if meteorism be present, may be completely obscured. Palpation as well as deep percussion[1] is restricted by the pain of the patient, as well as by prudence. Experience has often revealed to surgeons empyemata and abscesses which were at the point of rupture. Usually, however, it suffices for the diagnosis if we discover resistance over the cecum, or medially or laterally related to it. We may be fully assured of a firm limitation when a coarse but slightly painful tumor is found, and also when a large tumor, with a parallel retardation of the other symptoms, daily becomes smaller and more compact; yet these findings afford no guarantee against secondary perforation.

Vomiting.—Vomiting is due to reflex irritation of the peritoneum; in the malignant infections which are rapidly and widely distributed, it is often severe with the ejection of grass-green masses; in high-graded intestinal paralysis obstruction occurs under some circumstances, usually with the symptoms of collapse.

These forms are typical of acute diffuse peritonitis. In the circumscribed form the vomited material is rarely grass-green, but the vomiting is frequently severe and distressing, and often bears a certain relation to the intensity of the inflammation. Vomiting usually accompanies relapses of appendicitis, and especially when they run their course without purulent inflammation of the serosa; this symptom is met with, therefore, in many cases when all other signs of implication of the peritoneum are absent.

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[1] According to Weil, I understand by deep percussion that form in which the pleximeter or the pleximeter finger is pressed deeply into the belly.

Urine.—The urine varies, but these variations do not always correspond to the severity of the disease; in the severer cases, particularly those with high fever, it often contains albumin and indican; in children these, as well as aceto-acetic acid, are found much more frequently and often in decided quantities. Dysuric symptoms depend upon implication of the lower portion of the abdominal cavity; they may be temporarily observed when no other symptom points to the areas surrounding the bladder.

DIFFERENTIAL DIAGNOSIS

1. **The Differentiation of Perityphlitis from Other Conditions.**—In case we find a distinct tumor between the umbilicus and the anterior superior spine of the ilium the question of a fecal tumor comes into consideration. It must be remarked that this may be an accompanying symptom of perityphlitis, and, if accompanied by severe pain and vomiting, must always be borne in mind. A mere accumulation of feces also occurs here, but is much rarer in ordinary constipation than in local conditions which cause stasis, stenosis of the colon low down (tumor of the left flexure, cholecystitis with adhesion to the transverse colon), or stenosis at the cecum itself from old adhesions or thickening of the serosa, i. e., residues of a *former* perityphlitis; such a fecal tumor is moderately sensitive, movable, circular, usually compressible, and disappears after a bowel evacuation (as a matter of caution this had better be produced by enemata).

Neoplasms of the cecum or colon may, under some circumstances, also cause tumors in this region; these are also movable at first, are very coarse, non-sensitive or slightly painful. Here attacks resembling perityphlitis are very rare; paroxysmal marked stasis is produced much more readily; during the attack the diagnosis may occasionally be difficult on account of insufficient preceding observation.

The latter is also true of acute intestinal occlusion in this region, particularly that due to strangulation; invagination of the ileum into the cecum causes at first a characteristic tumor, later hemorrhagic dejecta; the condition cannot be readily mistaken.

Tuberculosis and actinomycosis of the cecum and appendix, particularly in the earlier stages, may lead to extreme differentio-diagnostic perplexity.

Besides appendicitis, other pathologic conditions produce painful attacks in the vicinity of McBurney's point, pain upon pressure, vomiting and fever, and these are: Cholelithiasis or cholecystitis, right-sided renal colic, particularly when combined with a dislocated kidney, inflammatory disease and tumors of the ovaries and of the tubes, gravitation abscess from the psoas. Here a careful history is of great value; not rarely the diagnosis can be made at once; a large liver and the respiratory movability of a tumor that may be present favors pericholecystitis; we must be cautious, however, in the consideration of moderate jaundice, since it also occurs in perityphlitis; gall-stones are rarely found in the feces, but, if present, they aid in the decision. This differential diagnosis is sometimes difficult or even impossible. If the kidneys are implicated, it will be evident from the well known manifestations of a dislocated kidney as well as the examination of the urine (nephrolithiasis). The question of disease of the female genitalia will usually be decided by a vaginal examination which should never be omitted in any case; naturally this is not always decisive, and especially when there is implication of the serosa of the pelvis in perityphlitis, or in case of simultaneous disease of the adnexa and perityphlitis. When considering gravitation abscess, the vertebral column and the sub-inguinal region should be carefully examined; the history is of value in this instance as well as in all others.

The most difficult cases are those in which no resistance is present and the pain can scarcely be localized, or where pain and resistance are present in a paradoxical area, as in the case of a very long appendix adherent at its apex; here the focus may be under the liver, in the pelvis, even upon the left side of the abdomen, and the diagnosis of appendicitis may cause the greatest perplexity. In all such instances, if the accompanying symptoms point to appendicitis, this affection should be first considered as it is relatively the most frequent. In late childhood it is of especial importance, as disease of the biliary passages and gastric ulcers seldom arise in these subjects; they occur at the end of the second decade of life, but do not often occasion symptoms that resemble appendicitis. From the thirtieth year upward the frequency of appendicitis diminishes (i. e., of the first attack!) and after the fortieth year the disease is rare. Hernia and internal incarceration must invariably be thought of: History and local examination including that per rectum!

2. **The diagnosis of the form of perityphlitis** is the most difficult part of this entire subject.

I must first emphasize that appendicitis in itself, particularly that form with but slight implication of the serosa and, at all events, without purulent implication, is very difficult to differentiate from periappendicitis. The affection begins with mild fever of brief duration, sometimes, however, with a decided rise in temperature and ileo-cecal pain; nausea and vomiting invariably point to implication, or at least to congestion, of the serosa; constipation may also be present. Local examination reveals pain upon pressure and in some of the cases a tumor, corresponding to the swollen appendix, globular in shape and often distinctly movable, sharply circumscribed, or apparently embedded in a firm mass—serous exudation in the serosa, in which case implication of the peritoneum may be assumed with certainty. This condition has long been recognized but was more accurately studied in the operations which Riedel performed early in his cases, and he explains it as an inflammatory, phlegmonous, or erysipelatous relapse of chronic appendicitis granulosa hemorrhagica. It is often the precursor of well developed attacks of perityphlitis. The diagnosis may be made with great probability when, besides the symptoms already enumerated, no sign of severe general implication, no special meteorism, no symptoms attributable to the lower portion of the abdominal cavity are present, and no large tumor has formed. It is well constantly to bear in mind that a perforative peritonitis may set in insidiously, and for this reason the patient must be carefully watched.

From what has been stated the difficulty of the differential diagnosis of the *forms of perityphlitis* may be imagined. Clear clinical diagnoses which include the anatomical condition of the appendix, the cecum, the peritoneum, etc., and besides convey an idea as to what may develop, can only be made in exceptional cases. For the present we must be content with a diagnosis which is made principally from the practical standpoint of prognosis and therapy; and here the burning question always is: Is the process localized, and will it remain so?

If no pus is present the attack as such may be looked upon as mild —with rare exceptions which shall at once be mentioned. I believe it likely that in the, fortunately, very rare cases in which a severe, diffuse, septic perforative peritonitis (with fecal stone always present) sets in with a stage of several days of mild fever, pain, and moderate vomiting— in these cases in the "prodromal stage"—nothing more is present than a simple appendicitis preceding gangrene. These are exceptional cases which we are unable to recognize by any method of our art. In general the law holds good: If there is no pus the case is mild, if pus is present it is serious, and when the pus is putrid, situated around or in the appendix, the condition is extremely serious, i. e., immediately dangerous to life; in the latter case perforation almost invariably takes place, and is usually due to fecal calculus; rarely does a putrid empyema form.

The question, therefore, arises: Can we diagnosticate the presence of pus, and can we recognize it as putrid?

Not long ago the answer to this first question appeared to be greatly simplified by the assumption that every perityphlitic exudate contained a purulent nucleus (Sahli); it *appeared* simplified—yet, from the practical standpoint of prognosis and from the treatment which was to be undertaken or rejected it nevertheless was not so—because the presence of pus is of very varying importance (compare page 544). But we are not always dealing with pus: We learn from the early operations, above all, those of Riedel, that there may be a serous, or sero-fibrinous, periappendicitis, and a recently inflamed appendicitis surrounding this.

Therefore, in an acute attack of appendicular disease, we are confronted by the question: Is pus present or not?

A fluctuating tumor appearing acutely in this region always indicates pus; a small tumor will by its resistance at least permit us to assume its fluid contents, although exploratory puncture is very rarely permissible; if any fluid be present, it is pus. But in the majority of cases we find firm tumors or tumors with more or less indistinct resistance. In positive tumors with firm walls an exploratory puncture may be made; but it does not in all cases show pus; this is only the case when the point penetrates to the purulent nucleus. In all cases of resistance and when positive walls are distinctly perceptible this operation should in my opinion not be undertaken, as the result is usually negative and it may disseminate the pus and feces; I once saw a hole made with a fine Pravaz syringe in the free wall of the intestine, which soon became gangrenous, as was determined by an autopsy shortly afterward conducted.

As is well known, the diagnosis of local suppuration is to-day based upon the presence of leukocytosis. Concerning this I possess no personal experience, and no conclusive opinion has been reached by other authors.

The result of this is that it is frequently necessary to consider the other symptoms of pus. I have previously mentioned that pus may be suspected in perityphlitis when fever, either continuous or remittent, lasts for a number of days, three to five or longer; chill at the onset without prolonged fever is an uncertain symptom; it may be produced, like a fever of brief duration, by a non-purulent appendicitis and by periappendicitis. Neither are violent vomiting and severe pain positive symptoms of pus. Empyema of the appendix may be diagnosticated if, with chills and high fever of long duration, a very circumscribed, round, excessively painful tumor appears in the right lower abdominal region.

Finally, pus infection, and at that the most serious form, the putrid, is present when the so-called symptoms of sepsis or collapse become noticeable.

Occurrences of this kind are rare; in the majority of cases the fever is less intense; nevertheless, pus may be present.

With a solid tumor, and even without a recognizable tumor, pus may form in the appendix or around it, and may even be putrid, yet the temperature remain normal, and pain not be excruciating. These latter cases naturally are rare exceptions—this is the only consolation they afford.

The absence of symptoms of pus does not therefore exclude the presence of pus; but when they are absent, when the picture of appendicitis simplex, as sketched above, and with or without periappendicular symptoms, is present, it may be excluded with a great degree of likelihood; notwithstanding this, we must be on our guard.

Pus in the appendix is, moreover, a very varying condition. There is a benign pus, particularly among the cases without perforation, and with clearly defined limits; i. e., it at once causes a very hard, moderately large tumor and, after a little time, probably by rupture into the intestine, disappears; but among the non-putrid suppurations there is also a very malignant form, usually seen in perforation, but also, although very rarely, without this. Here also the rule is operative: The malignancy, i. e., the tendency to rapid progression, is often manifest from the typical symptoms, but it cannot be excluded when milder symptoms only are present. That the relative position of the abscess and cecum is of importance has already been stated.

How may we recognize putrid infection which, in the majority of cases, indicates fecal calculus perforation? The symptoms of septic collapse, even when only indicated, must always arouse this suspicion; in a successful exploratory puncture a positive diagnosis can at once be made by the sense of smell. With chill, continued high fever, very severe pain, and especially with pelvic or other severe local symptoms (marked meteorism, singultus), with albuminuria and very marked indicanuria, this must always be thought of, particularly, however, when these decided symptoms are contrasted with the absence of tumor; with mild symptoms it cannot be positively excluded, *yet it is rare.*

Comprehensively the following may be stated:

1. As a rule, vomiting, ileo-cecal pain, a moderate fever not lasting longer than three days, and very circumscribed or indistinct resistance are in favor of appendicitis simplex (inflammatory relapse of appendicitis granulosa hemorrhagica, Riedel), with non-purulent implication of the serosa. A moderately severe chill accompanies this condition; very severe pain upon palpation does not disprove it.

2. As a rule, the same symptoms with protracted fever reaching to 102.2° F., and with a larger, coarse tumor indicate purulent, non-putrid, circumscribed periappendicitis; moderate meteorism occurs at the onset, but then disappears.

3. As a rule, an extensive tumor between the umbilicus and the anterior superior spine of the ilium with severe symptoms (chill, frequent vomiting, moderate meteorism), but from the third to the fifth day, and synchronous with the fall in temperature and cessation of the vomiting, a certain but limited decrease in size favors perforation of the appendix but with a tendency to solid encapsulation of the abscess which is for the most part putrid.

4. A sudden onset with fulminant symptoms, extensive painful meteorism, pelvic disturbances, and the absence of distinct resistance, awakens the fear of an insufficient limitation of a perforative peritonitis. Signs of so-called septic collapse, even the slightest, are, however, the positive evidence of this condition.

5. Retrocecal and retrocolic symptoms (movements and position of the leg are to be observed, pain in the posterior lumbar region) always favor pus, which need not, however, be putrid.

6. Chills, high fever (later declining), excessive vomiting, and **very severe** *pain with a circumscribed round tumor from the onset favor em-*

pyema. In the majority of cases this is not putrid; for this reason encapsulation of the empyema is usually firm.

7. As a rule, when several relapses occur, perforation by fecal stone will have been most severe in the first attack; if, therefore, an attack suggestive of perforation have already occurred, the relapses are usually milder; inversely, if attacks of non-perforative periappendicitis begin with an appendicitis granulosa and stenosis, usually mild at first, and gradually becoming more severe, gangrene and perforation eventually occur; the preceding attacks are then usually a certain guaranty of preceding adhesions.

8. **Exceptions to all of these rules occur, and in all directions.** *The more serious are those in which* **malignant affections set in with mild symptoms.** *The gravest condition is that of ichorous empyema and ichorous perforating abscesses with mild initial symptoms and sudden rupture into the free abdominal cavity.* **Neither occurrence is very frequent.** *Much more often is a tendency to secondary distribution gradually revealed by more severe and more distributed pain, higher fever, and more rapid pulse, vomiting and mild meteorism which will be apparent to the careful observer. Usually with secondary distribution we are dealing with mild or even moderately severe cases of progressive purulent peritonitis; almost invariably these are due to perforation from a fecal calculus. Abscesses at the medial side of the cecum are most prone to show this tendency.*

GENERAL REMARKS REGARDING THE COURSE OF PERITYPHLITIS

According to various comprehensive statistics, particularly Sahli's collective investigations, it may be stated that acute perityphlitis without operation is fatal in from 4 to 9 per cent. of all cases, and that relapses occur in about 20 per cent. of non-operative cases.

Relapses.—These relapses must occupy our attention for a moment. They occur in perforative, as well as in non-perforative perityphlitis, but even according to previous statistics they are much more rare in the former; when we consider the milder "relapses" of "appendicitis granulosa," the greater proportion of relapses in the relatively benign forms will become apparent.

The first relapse generally occurs in the first, less often in the second year, and much more rarely thereafter.

In the perforative forms, the first attack is usually the most severe the relapses are benign, and occasionally show a gradually decreasin severity. The earlier, however, the first relapse occurs after the firs attack, the more certainly must we reckon upon its malignancy; one tha occurs after a few weeks is not infrequently more serious and occasionall even fatal. These relapses may be caused by various conditions: By propagation to the abdominal cavity of the pus which has not yet dis appeared (delayed progressive purulent form, malignant), to the retrocolic tissue, etc.; by the sudden occlusion of a perforation into the intestine before the pus has fully discharged; by induration and adhesions which hinder the passage of feces. Accordingly the character and the prognosis of the attack vary greatly.

Chronic appendicitis without fecal calculus, and with or without stenosis, frequently causes "relapses" of gradually increasing severity in which implication of the peritoneum is at first doubtful, but may be later more distinct; finally, pus forms and meets with adhesions previously for a long time present. But in these cases without fecal calculus, even when the first (*distinct*) attack produces periappendicular pus (usually without gangrene, therefore without perforation), the course, according to the experience of Riedel, is generally favorable: Absorption or rupture—induration for a varying time, occasionally only amounting to days—stenosis or, what is most favorable, complete obliteration of the appendix and entire recovery. In rare cases, naturally, the abscess persists, possibly with secondary complications (descent, slow rupture into an inconvenient point, for example, the bladder, venous thrombosis, etc.)—or without this, with chronic invalidism associated with pain, occasional fever, and dread of eating. Or, indistinct febrile attacks come on even when the abscess has disappeared; some time ago, in one of my cases of this kind, a torpid flat ulcer was found in the thickened appendix which was everywhere adherent. Not infrequently attacks of gradually decreasing severity are observed for years; here pus is no longer present, the adhesions and indurations gradually disappear; the nature of the difficulties produced by these conditions we shall soon refer to.

Long-continued abscesses usually give the patient no rest; however, long periods of latency (over a year) occur, exceptionally even with putrid abscesses, and under some circumstances with sudden, severe, peritoneal exacerbations.

I recently observed an instructive case of this kind.

A robust young man, 22 years of age, had two years previously a moderately severe febrile inflammation of the cecum which "healed completely"; since that time he has now and then suffered from indistinct drawing pains in the right lower abdominal region; lately he had a mild relapse, with moderate fever lasting a day, violent pain, constipation, and nausea. He was a strong, well nourished man. A tumor about twice the size of a plum, compact, round, and slightly movable, was found below McBurney's point. Slight pain upon deep pressure aroused a suspicion of pus; the conditions were otherwise negative. *Operation: Old non-putrid abscess around a perforated, otherwise markedly thickened, appendix; fecal calculus.*

If abscesses, after a prolonged duration, finally rupture into any organ, it may happen that, in the meantime, rigid cavities will have formed which, even though communication is open, do not heal; therefore the result is invalidism. But even old, closed, rigid wall cavities may form

It is frequently noted in the pelvis, sometimes originating from immediate surroundings, and sometimes descending from the appen or from the colon. Another point of preference is the vault of the (phragm: Here the intraperitoneal subphrenic abscess is usually situa particularly if it contains gas; pyopneumothorax subphrenicus is usu to the left of the suspensory ligament of the liver, but also occasion to the right of it, and rarely bilaterally. All of the organs bordering the subphrenic region are implicated in the development of these absces The stomach, the liver (that is, the biliary passages), the duodenum, pancreas, the colon, the kidneys, the spleen, the ribs and vertebral colu the diaphragm from the pleura, the lungs and pericardium. The majo of abscesses originating from the biliary passages are situated below liver; frequently they extend almost to the ileo-cecal region, and n cause great difficulty in the diagnosis; if, to aid us in our decision, exploratory puncture be risked, pus admixed with bile, or frequently o pus, is found. Abscesses of the colon, often of obscure origin, are oc sionally situated here, or they may be opposite the ascending colon and the flexure; they are usually circumscribed and prone to be chronic.

The same is true of these abscesses as of the periappendicular for They may show firm encapsulation and, particularly when small, n remain latent for a long time; according to their position they may rupt in any direction, through the abdominal wall into a hollow organ, es cially into the intestine or into the bladder, or through the diaphra and ultimately into the lung; they may by erosion force their way i a solid organ, as, for example, perigastric abscesses and abscesses of biliary passages into the liver; by rupturing into the free abdominal cav they may immediately produce a diffuse peritonitis or develop a p gressive purulent inflammation. Finally, they may readily lead to p mia, particularly by septic venous thrombosis. If the abscess is small a insignificant, situated deep and not susceptible to examination, a cryp genetic septicopyemia may develop clinically.

To enter into further details is at this point impossible.

PROPHYLAXIS AND TREATMENT

Prophylaxis.—As to the prophylaxis of acute diffuse peritonitis, m ern asepsis and antisepsis in the hands of the surgeon and gynecolog are powerful agents to prevent the development of this affection af operations upon the abdomen, in the treatment of abdominal injur and during labor. The situation is more complicated when we consi the numerous pathological conditions which expose the peritoneum to flammation from its surroundings. Here it would be well if we co decide in the individual case whether the process has been so extens as to produce inflammatory conditions or those that lead to danger rupture, and also whether an *invasion into the abdominal cavity is likely.* But, unfortunately, this is seldom possible.

In round ulcer of the stomach and of the duodenum and in enteric ulcer this is never the case, and even in the pancreas we are far from accuracy in clinical judgment. In gall-stone colic with fever, or even with chills, serious consequences may more readily be prevented; appendicitis and perityphlitis have just been accurately described. In incarcerated hernia, in strangulation and in invagination, surgical treatment which prevents peritonitis plays an important rôle. But upon this subject, and also the diseases of the sexual organs, we shall not now enter.

In not a few of these conditions the aid of the surgeon is needed before there is any thought of a diffuse peritoneal complication. But even where this has been previously impossible, the family physician or the internal clinician should consider himself honored by inviting the surgeon in consultation, should he be at hand and the patient permit it, and in obtaining his opinion so soon as a diffuse peritonitis seems threatening, or even before it has developed. Thereafter the case belongs to the surgeon.

If the physician, however, finds an acute diffuse peritonitis already developed, the question of treatment is directed primarily to the point of origin; whether, and how, it may be attacked is the privilege of the surgeon to decide.

In many cases, from a theoretical standpoint, we must strictly discriminate between bacterial and chemical peritonitis. I have previously stated of the latter form of peritonitis that it stands or falls with the bacterial focus of origin; therefore this should be immediately removed, when the rapid disappearance of the diffuse symptoms may be expected. Considerations of this kind will, under some circumstances, influence the surgeon as to his manner of procedure. But it is neither my object nor my function to enter into more minute details in regard to this. Practically, it is evident from the foregoing that the clinical differentiation between malignant bacterial and benign chemical peritonitis (peritoneal irritation) frequently occasions the greatest perplexity.

Operative Measures.—Therefore, according to our present standpoint of therapeutic knowledge, surgical relief is to be sought in all cases of acute diffuse peritonitis except those in which decided symptoms of severe peritoneal collapse have already appeared. Mild and moderately severe clinical pictures of this nature are, as a rule, not positive contraindications for an operation; but subjective judgment as to the patient's powers of resistance, particularly the condition of the heart and the vasomotor system, as well as the severity of the other toxic phenomena, leaves wide room for doubt; definite rules cannot be established. It must always be borne in mind that, as a rule, operation at first intensifies the collapse.

On the whole, a comparative estimate (in so far as this is possible) of the results with and without operation proves that in perforative peritonitis, if operation is very early, i. e., inside of twelve, or, at most, twenty-four hours after perforation, there is slightly more, but, nevertheless, greater hope of recovery than without operation. Those cases are naturally the most favorable in which peritonitis has not yet developed, provided that up to the moment of operation there is no peritoneal collapse. If the latter occurs, the prognosis in laparotomy is exceedingly grave and, therefore, it is rejected by most surgeons.

A relatively unfavorable prognosis after operation is presented by those cases in which previously more or less localized relapses of progressive peritonitis have occurred, after which a severe diffuse picture is suddenly presented, and also in those cases in which, combined with a circumscribed peritonitis, the indistinct picture of diffuse peritoneal irritation had some time previously existed, this having abruptly changed to a severe diffuse inflammation; here, as a rule, there is a massive secretion of pus, widely distributed. These occurrences are usually preventable by proper surgical treatment of the preceding circumscribed peritonitis (see below).

The conditions are likewise unfavorable for surgical intervention i
peritonitis to which a septic condition has been added, or in which thi
already coexisted, as is so frequently the case in the puerperal form
Here also, for good reasons, the surgeon refuses to operate.

For the not infrequent cases where either a diffuse peritonitis is certai
—its origin, however, not being quite clear—or in which the differenti
diagnosis between peritonitis and strangulation or the like cannot
readily made with certainty, early surgical treatment is generally advisabl
It is a serious error to confound this condition with coprostasis, uremia
peritonism, although I can easily imagine isolated cases in which t
physician, and also the surgeon relying upon his asepsis, might with
uncertain diagnosis assume the responsibility of deciding upon an "
ploratory laparotomy."

These criticisms, which in part extend into the realm of surgery
may be permitted to make because they indicate the cases in which
internal clinician or the house physician would make a gross error
to consult a surgeon, and because they show that the surgeon's aid
usually needed early, and that, to avert danger, laparotomy must spee
be performed.

From a purely practical standpoint, however, the conditions are
that where the surgeon may be readily obtained his opinion should
sought in *every* case in which acute diffuse peritonitis appears to be
tain or likely, or even possible. This cannot be emphasized too stron
If he can be reached only with difficulty, the internal clinician hi
must weigh the possibilities and decide whether or not the aid of
surgeon is necessary.

Internal treatment must be resorted to in those cases in which o
tion is rejected and those in which surgical relief is either unobtai

or cannot be secured at the right time. These are almost all lost, but none of them present an absolutely bad prognosis; this must be borne in mind. We may see most remarkable recoveries from the severest clinical picture, even of peritoneal collapse, and particularly in appendicular peritonitis; I have known the symptoms to disappear after rupture and recovery finally to take place in cases where, without rupture, the heart would have held out but a few hours longer. Such outcomes are unfortunately rare, *but they must stimulate the physician in each individual case to continue the battle to the end.*

Internal Treatment.—We possess no radical internal treatment of peritoneal suppuration, i. e., antitoxic or bactericidal, which promises success.

Two principal indications are to be met by the *internal treatment* of acute diffuse peritonitis: Absolute rest of the areas surrounding the focus of origin and also of the entire abdomen, and the maintenance of the strength of the patient as long as possible. With this, too, is the important consideration of relieving the sufferings of the patient.

The first indication calls for absolute quiet, avoidance of shock to the abdomen, therefore, above all, the prevention of vomiting, the stopping of all, or nearly all, food by the mouth, the arrest of intestinal peristalsis, psychical repose.

The necessity for absolute quiet is often confronted by the question of moving the patient to a hospital; here, under all circumstances, the individual case must be considered; the issue cannot be discussed from a general standpoint. The vomiting is at once somewhat favorably influenced by opium or morphin; if this, however, should not be the case careful lavage of the stomach with a cool fluid may be employed soon after a meal, particularly if we suspect a complicating ileus or a perforation from ulcer, and in other isolated cases in which we are under the impression that continued severe retching will be more injurious than the perturbation produced by the stomach-tube. In conclusion, I cannot advise the general employment of the stomach-tube, as some authors have done.

Food by the mouth is to be stopped at the onset; but, in the course of the affection, circumstances will not seldom compel us to return to this—naturally when improvement begins. The first food that may be administered is meat juice freshly expressed (by means of a meat press), then cool bouillon with the addition of Plasmon, Leube-Rosenthal's meat solution and the like, following this milk, cooled by ice, all to be given in teaspoonful or tablespoonful doses; diluted wine or cognac is permissible. The subsequent stage of beginning convalescence I shall pass over. Administration of water by the mouth should only be stopped in the severest cases; small pellets of ice may usually be swallowed, unless this increases the vomiting which may occur.

Administration of food by the rectum is by no means immaterial; as is well known, it may stimulate peristalsis in the entire colon. If we are forced by the failure of strength to employ this method, I advise only enemata consisting of two eggs with 1.0 sodium chlorid to each egg (sodium chlorid 20.0 to 200, 10 grams or one small teaspoonful per egg). If there be temperature, about 15 cm. of this solution is to be cautiously injected high up, or milk, perhaps with an egg and about 5 per cent. of sugar. Anything else will readily irritate.

Water is particularly valuable for conserving the strength, and much more indispensable than food. *The organism must not thirst!* According to the case and how water is borne, it may be given either by rectum or subcutaneously (in the thigh) in as large quantities as possible (in the latter case sterile physiologic salt solution of the temperature of the body, about 200 at a time, or by continuous infusion).

Of course, if the patient becomes chilly he should be made warm; that is, external heat should be applied.

To meet the indication for complete rest of the intestine, the use of opium in large doses, perhaps of morphin, immediately at the beginning of the disease, is recommended. It is true that the bowel rests in consequence of the peritonitis itself, although it is questionable whether this is invariably the case, for example, in those cases associated with diarrhea; at all events clinical observation proves that rest of the intestine is promoted by opium; the decisive significance of which is obvious, and, in comparison, the deleterious effects of its energetic use are unimportant. In this connection it is urged that it increases constipation, masks the course of the disease, and acts unfavorably upon the strength, particularly upon the heart. In diffuse peritonitis I believe these views to be without weight. Constipation in diffuse peritonitis is almost always unimportant, particularly when we consider the abstinence from food; what, however, opium may be said to conceal is not quite clear to me, since the decrease of pain and vomiting while other severe symptoms persist cannot deceive the physician, and the somnolence produced by drugs can lead to no diagnostic doubt regarding the true condition, provided, naturally, that actual intoxication is avoided; finally, as regards the unfavorable effect of the alkaloids of opium upon the heart, this, as is well known, is very slight and is readily compensated for by its favorable effect in diminishing pain and vomiting.

Treatment by opium is deleterious to a high degree when it is the resource of those who cannot decide upon an operation; here the anodyne and stupefying effect of the remedy is a source of great danger. For the critical physician, however, who objectively grasps the actual state of affairs, no such danger exists; perhaps he may prefer to let the patient bear a little pain for some time in order to win his consent to the life-saving operation! I can readily imagine such a state of affairs.

In a treatment with opium the fact must be borne in mind that the drug should be given as early as possible and in decided doses; with continued employment its paralyzing effect upon the intestines soon ceases; often in the subsequent course of the affection, and according to the condition of the patient, the treatment must be at times suspended. Therefore, at the onset a dose should be given hourly or every two hours, later every three hours, and then even more rarely, and when aggravation occurs increased doses of from 0.03–0.05 of pure opium or the extract of opium, or six to ten drops of tincture of opium are to be administered by the mouth. Vomiting once or twice (pellets of ice to be given) need not cause the suspension of this method of administration. If vomiting increases, the drug should be used by rectum (enemata or suppository) or morphin, which unfortunately does not have the same effect, by subcutaneous injection of about 0.01 with the intermediate pauses mentioned above. Naturally, when such doses are employed the symptoms of poisoning must be watched for, particularly those on the part of the sensorium and the pupils, and we must remember that the pupils contract under moderate toxic action; when it is very severe they dilate. Moderately narrow pupils do not contraindicate the continued cautious use of the drug. I have never seen an instance of the sudden cumulative effect of the remedy, which indicates that after a period of non-absorption several doses have suddenly been absorbed. It is possible that this occurrence as reported is due to confusion with peritoneal collapse and loss of consciousness; however, these remarks are not to be looked upon as a criticism of the reports just referred to.

Morphin acts upon the mind and the sensation of pain to at least the same extent as opium, upon intestinal peristalsis and vomiting less intensely; it is therefore an imperfect substitute for opium, which we are only to employ when nothing but the subcutaneous injection remains, or when we wish to effect a rapid action, as when pain is the most prominent symptom in the case.

Other substitutes are only to be considered when, on account of repeated attacks of vomiting, which are of very serious import, or the solution of adhesions, or the distribution of the generators of inflammation, trial is made of other drugs. Tincture of iodin in drop doses in a little water is an old remedy which is occasionally very successful; cocain is useless; only gastric lavage remains which in such cases is advisable; naturally but small quantities of water and very slight pressure are to be employed.

Of cardiac and vasomotor remedies in collapse, above all in *threatening* collapse, camphor and caffein, subcutaneously injected, are recommended.

In very isolated cases, particularly when the fever disappears, the pain and meteorism have become less, yet feces are not discharged, and there is marked dulness of the descending colon, the indication may arise to evacuate this organ carefully. I should not do more than use about [illegible] grams of olive oil by enema. The careful observation of the state of the bladder and, with a prolonged duration of the disease, the prevention of bed-sores, must always be borne in mind.

CIRCUMSCRIBED PERITONITIS, PARTICULARLY PERITYPHLITIS

We must consider also the therapeutic indications in perityphlitis. First, because it is the best guide to the points suitable for consideration, partly because it is the more frequent affection, and also because the consideration of all other forms of circumscribed peritonitis ranks in importance with the treatment of all the organs of the abdomen and their surroundings.

PROPHYLAXIS

The prophylaxis of perityphlitis up to the present time is somewhat chaotic. The apparent increase of this disease, particularly in the United States, has led to the supposition, which is perhaps most reasonable, that here hasty eating, insufficient mastication, and constipation are certain causative factors; the conclusion is obvious that persons who from a fear of appendicitis eat slowly and masticate thoroughly may thus become hypochondriacs. It is much better to relegate to general hygienic strictures all advice of this kind.

We are dealing with tangible objects when we consider fecal calculi which may certainly remain in the appendix a long time before the become dangerous, and also when we consider chronic appendicitis an its exacerbations, the "relapses" which we have already described.

Up to this time the latent fecal calculus cannot be diagnosticated, no even with the X-rays; here, therefore, is no question of prophylaxis; if i were possible to recognize it, and considering its danger and the preser certainty of surgery, no doubt it would be well to remove it with i enclosure, the appendix.

Here the indication would be simple. Much more complex are t conditions in that other precursor of perityphlitis, chronic appendiciti here the diagnosis is clear if the "relapses" are correctly considere but it is not at all certain that mild "relapses" lead to purulent pe appendicitis, or even to necrosis and perforation: The danger of chro appendicitis is far less than the danger of fecal calculus. The questi resolves itself into this, whether in pure appendicitis we should oper to prevent a purulent perityphlitis, and this is certainly worthy of seri reflection. We shall consider it in common with the operative treatm of perityphlitis.

TREATMENT

In the treatment of perityphlitis which has either been diagnosticated with certainty or with a great degree of probability, in general the following facts are to be weighed: Under internal treatment, i. e., without recourse to surgery, from 70 to 80 per cent. of all cases entirely recover, a number which is large but which, naturally, does not comport with our therapeutic endeavors; in the treatment of these cases, that therapy suffices which has for its Alpha and Omega the keeping of the intestines and the entire abdomen absolutely at rest. Of the cases that remain, no doubt nearly all might be cured by timely operative measures; in the majority of these cases, too, a surgical operation might be performed if the right time were chosen, and the patient were carefully examined and watched. In a small proportion of these cases, however, it is done too late, partly because, without fault of the physician, and without prodromes, there is a sudden perforation into the free abdominal cavity, but partly also because the case was an apparently benign one which gave hope of recovery by internal treatment yet it suddenly became dangerous, or because at the beginning it appeared to improve and a sudden fatal relapse occurred. These last two categories will always rest as a weight upon the conscience of the physician.

From the preceding observations it will be noticed that the most serious cases and those requiring most speedy operation are the following: 1. Perforation into the free abdominal cavity; 2. Larger abscesses which usually perforate, and are characterized by an increasing tumor and a tendency to further enlargement, a "progressive condition," noted particularly upon the inner side of the colon; 3. Empyema without marked adhesions, especially the putrid form. In contrast to these are the relatively mild conditions: Non-purulent periappendicitis; small non-perforative abscesses with solid adhesions, which terminate partly by absorption and partly by rupture in the direction of least resistance, i. e., toward the intestine; finally, mucus and sero-purulent, moderately virulent, non-putrid collections of secretion in the appendix.

These conditions very rarely lead to immediate danger; but during healing they may produce strictures and kinking, and by relapses develop a malignant character—although this is very rare.

Midway between these two categories, in regard to spontaneous cure, are the moderately large abscesses which sometimes show fine perforation and sometimes do not perforate, and in which the danger depends upon the continuance of preformed and fresh adhesions, upon the virulence of the pus, and upon the promptness of its rupture in a favorable locality. These abscesses are rarely absorbed. They form a large proportion of the total number of cases of perityphlitis.

I repeat what has already been said elsewhere in other words, in nerves. These conditions are occasionally of vital importance, especially in the female during labor. To this must be added the instinctive repugnance of man to mutilation, which in a mild illness makes it difficult for him to consent to operation for an affection which is usually only slightly dangerous.

The relegation of all forms of the disease to the knife of the surgeon will, therefore, I am firmly convinced, never be possible; on the contrary, on account of the rare dangers which, however, can never be completely excluded in the individual case in the milder forms of the affection, the question arises in how far is operation necessary.

On this point opinions are far asunder, which is only to be expected. The perplexity which assails us in an exact discrimination of the cases is very great; aside from a certain tenacious adherence to the old, which to some extent still shows itself in internal medicine, and aside from the very prevalent error of the surgical author in considering as the normal whatever he is able to do for himself, each position has its justification.

The question to be put in the "intermediary" cases is a double one: 1. Which cases are to be operated upon during the attack? 2. Which are to be operated upon after the attack? In regard to the first, many authors are of the opinion that operation is to be immediately undertaker when pus is present or when it is likely to be present. Here we mus face the previously mentioned diagnostic difficulties based on the genera assumption that pus always accompanies acute, painful, febrile tumor: a position that can no longer be maintained; for pus may be absent whe we have quite high fever, severe vomiting, and a distinct, painful tumo Besides, the catchword "pus" by no means embraces everything; the dar gerous character of a periappendicular pus focus varies according to tl position and the length of existence of the adhesions, according to wheth or not perforation takes place, whether or not it is putrid—and not on the local but also the general symptoms are of significance; they m manifest severer, or milder, conditions than those present. Finally, p may be absent in one "relapse" and appear in the next one.

I advise, in general, in those cases in which pus may be assumed a [illegible] of the tumor, when it increases in size [illegible] with a [illegible] of the fever and i [illegible] general condition and pulse, all of which point [illegible] with a surgeon a [illegible] When the previo [illegible] will rarely be [illegible] will almost alv [illegible] with mode [illegible] will be undertaker good time. For this, however, it is necessary **carefully to watch** the patient, and this, in fact, should be done as conscientiously as possible; where, however, it is impossible, the list of operative cases will be enlarged. *In these intermediate cases the question whether or not the patient can be watched has a strong bearing upon the question whether or not we shall operate in the attack.*

In many of these cases, if we do not operate during the attack, the question of operation may again arise in a few weeks, and invariably if there is a suspicion of persisting pus.

Operation after an attack has advantages and disadvantages in contrast to those of operation during an attack. I shall not enter upon the pros and cons, as this is a purely surgical question. Whoever rejects operation during the attack must naturally take account of the risks. Here, too, watching the patient is specially important.

Neither can I answer the question as to the method to be chosen, whether immediate removal of all the pathologic conditions, or the opening of the abscess and the subsequent extirpation of the appendix.

It is not my function to discuss the surgical indications in slowly progressing cases with the appearance of new pus foci in the abdominal cavity, in the belly wall, or in the pelvis behind the colon (examining the lumbar region!), in the subphrenic tissues, or in the pleura. But it is the duty of the physician on the slightest sign of complication of this kind, for which search should be made in all cases, to call in the surgeon. Prior to this, however, these cases have usually come under surgical treatment.

Non-operative Treatment.—I have previously stated that the Alpha and Omega of treatment in those cases of perityphlitis and also of appendicitis itself in which an operation is, or appears to be, unnecessary, or is not permissible, is to keep the intestine and the whole abdomen quiet.

The indications are primarily the same as in diffuse peritonitis: Rest (great care in transportation to a hospital), no administration of nourishment by the mouth at the onset or even later, nor, if possible, as long as inflammatory symptoms continue; if these retard, or the condition of the patient shows that the strength must be increased at any cost, the same dietary mentioned upon page 561 should be employed. The intake of water by mouth must be moderate, always small quantities only at a time, and perhaps only ice. At the onset food should never be given by rectum; nor should enemata then be given to empty the rectum; nevertheless, in protracted cases it may be necessary to risk nutritive enemata when that mentioned upon page 562 may be used. In such cases the subcutaneous injection of water may also be necessary.

The application of ice or cool or lukewarm Priessnitz compresses may depend upon the wishes of the patient. At the onset hot cataplasms must be strenuously forbidden.

Here also opium is the sovereign remedy; it must be employed in *all* cases of appendicitis and perityphlitis, provided it does not induce vomiting; in this case suppositories are to be resorted to, and only when these are not well borne should morphin, which does not act so favorably, be used as advised upon page 563.

In the old so-called typhlitis stercoralis it was found necessary to vindicate the position of opium, and this is the case even to-day. The same deleterious effects are alleged as in the case of acute peritonitis, and it is maintained that by increasing the stagnation of feces it induces a relapse in appendicitis (Riedel). Particularly is this true in perityphlitis, which frequently shows a retardation of all the symptoms after the first fecal evacuation, and invariably leads to the assumption, similarly as in stercoral typhlitis, that any evacuation of the intestine as such acts favorably, and that stagnation of the feces therefore acts unfavorably, so that we dare not increase the latter condition.

It must be admitted that those who, in milder cases of acute perityphlitic tumor (therefore, those in whom a cecal stasis of feces was previously looked upon as the cause of the disease), risk a decided dose of castor oil occasionally see all the symptoms disappear when the oil has had its full effect; in such cases it is questionable whether we are not dealing with a relapse of a chronic appendicitis (without periappendicitis!) and secondary fecal tumor from intestinal paralysis, or whether the appendicitis was not favorably influenced by the evacuation of the large intestine. I can readily imagine that the future will see a purgative treatment of chronic appendicitis and an energetic purgative treatment for acute relapses, *provided that, in the individual case, the presence of a fecal calculus in the appendix and the presence of inflammatory products, etc., of the appendix may be excluded with absolute certainty, and also that we may exclude a beginning invasion of the serosa; to-day we have not advanced so far!*

In every case, however, of fecal calculus, or when, without fecal calculus, products of inflammation appear upon the surface of the serosa, or when they are extending to this tissue, or have already formed adhesions, the stimulation of peristalsis is a grave danger, and absolute rest of the intestine is the principal indication.

And, since it is as yet impossible to exclude these cases with certainty in acute disease accompanied by pain in the right iliac fossa, purgative treatment in cases of this kind should not be resorted to. According to the present status of diagnosis, we must reject this form of treatment, not only by the mouth but, with very recent inflammation, for even a longer time by rectum.

Opium, on the other hand, is even more valuable here than in diffuse peritonitis: for the salutary intestinal paralysis due to inflammation in circumscribed perityphlitis implicates only the nearest and most adjacent parts: The cecum, a portion of the colon, perhaps the lowest portion of the ileum; it is, however, necessary to prevent peristalsis in wide areas, to increase the localization of the poison, and to moderate the propulsion of the contents of the small intestine toward the lowest portion of the ileum and cecum. This latter point appears to me to call for a little reflection.

Therefore, in every acute case, as in the case of acute peritonitis, the treatment should be opium by the mouth in large doses, abstention from food, and absolute bodily rest in so far as this admits of the transportation of the patient; with repeated vomiting, suppositories, possibly morphin subcutaneously.

When there is stubborn and violent vomiting, particularly, however, when there are symptoms of intestinal obstruction, subcutaneous injections of morphin are indicated, as well as when very severe pains are present which necessitate speedy relief; here this remedy may also be combined with opium.

In the opium treatment, however, it must be borne in mind that prolonged, continued doses of opium rapidly lose their effect upon intestinal peristalsis, as is usually evident in the treatment of chronic diarrhea; therefore, it is advisable that after the administration of two doses, smaller doses should rapidly follow or the treatment be interrupted if the circumstances at all warrant this (fever, vomiting, pain, meteorism, decrease in the size of the tumor); with the slightest aggravation the dose should be immediately increased. In severe cases in which operation is declined, or in which it is impossible, this drug under some circumstances may also be given for weeks with remissions and intermissions, and in these patients we will have an opportunity of observing the highly ameliorating and localizing effect of the remedy.

In severe cases, the periodic administration of mild narcotics, even when the pupils are moderately contracted, is permissible, and is recommended; naturally, in such cases there must be careful and experienced watching continuously. Where this is impossible the doses must be smaller.

The most serious misuse of this remedy should, however, be strenuously avoided; it consists in this, that physician and patient are content with the amelioration of pain instead of deciding upon an absolutely necessary operation!

In regard to local derivatives in the acute stage, leeches alone can be considered, as is also stated by Nothnagel; I have seen amelioration from their use (which, however, may also be induced by opium or morphin), and perhaps lessening of the vomiting, but scarcely more than this. With the previously mentioned indications they may occasionally be applied in very robust individuals, at least four at a time.

If the perityphlitis ameliorates, the local application of cold should be followed by moist heat, and this, if possible, should be continuously used until the resistance disappears; food may be very cautiously administered according to the indications upon page 561. Drugs had better be avoided. It is important in this stage that patient and physician wait for the first fecal movement, in case this has not yet appeared.

I saw two serious relapses which occurred after the disappearance of fever for several days and with an almost painless limited tumor and otherwise entirely normal abdomen, after the cautious injection of a small, lukewarm enema, and since then I leave the rectum alone as long as possible, all the more so as I cannot remember having seen a relapse which was due to constipation. Now and then accumulations of feces in the colon are uncomfortable for the patient, although here a psychical factor, i. e., the knowledge that the feces have not been voided for such and such a length of time, is coöperative; but this, too, is exceptional: constipation of eight days and longer is usually well borne. In the case of tenesmus it is self-evident that the patient must be cautioned against employing the abdominal press, but this caution is often neglected. Following the first spontaneous and very copious fecal evacuation I have observed renewed sensitiveness upon pressure and fever. Not a few patients report a "wound" sensation upon the right side low down. This indicates the need of care. In case of necessity, after the first evacuations have occurred, emulsions of oil or castor oil may later be given by rectum.

With a tumor still decreasing in size, with continued fomentations, the diet must be strict (gruels, eggs, meat juice, beef tea, cooked cereals): the patient must be kept in bed until all symptoms have disappeared. If absorption of the tumor is delayed, if, moreover, there is not the slightest indication of pus after two, three or four weeks, the time comes when the patient can no longer be kept in bed, and more solid food cannot be denied him. At all events, he must not leave his bed until spontaneous pains have disappeared, until the freest movement of the right leg is painless, and the pain upon pressure, except an indistinct, dull sensation, has disappeared; then an abdominal bandage (which should also be worn while in bed between the fomentations) must be worn; it should firmly support the parts and protect them; the patient should remain in the recumbent posture as much as possible, particularly after meals. The bowels must be carefully regulated, above all, by the unsurpassed enemata of oil. There should be no sudden movements, no bending, etc., for the first period.

Subsequently and for a long time the physician should watch the abdomen and the fecal movements. If the latter are disturbed he must ascertain whether intestinal torpidity, mechanical obstructions by induration and adhesions, or new inflammatory irritation is the reason. In the latter case absolute rest, abstention from food, and, perhaps, according to the intensity and duration of the symptoms, opium, are indicated, but by no means a purgative. If intestinal torpidity or obstructions due to induration, etc., are present, mild purgative treatment is advisable, but in the latter case we must be careful, above all, when infectious perityphlitic residues cannot be excluded with certainty. Under these circumstances it is obvious that purgative enemata are often to be preferred to drugs by the mouth.

The treatment of persistent indurations with various baths, above all, with mud baths, fango, poultices, etc., is, as is well known, often effective. Satisfactory results are doubtless now and then seen from massage, but usually it is productive only of harm, and as, up to the present time, it is impossible to foreknow the cases in which it will be useful and certainly do no harm, the method is too risky and should be rejected; still more is this the case with horseback riding, bicycling, etc., which are now and then advised.

For the rest, many conditions which are here considered will also be discussed in the description of chronic peritonitis.

II. CHRONIC PERITYPHLITIS

By J. BOAS, Berlin

As is the case with acute perityphlitis, chronic inflammations of the vermiform process have multiplied with the changes produced in this realm of disease by surgical intervention. By its agency our formerly vague conceptions of the nature and the course of inflammatory processes have assumed definite shape, and are based upon exact knowledge. Laparotomy has solved numerous problems which, up to very recent times, still clouded the teaching of perityphlitis, and solved them much more completely than did necropsy.

Internal medicine with its greater conservatism followed the paths marked out by laparotomy only slowly and cautiously. But the impression produced by surgical successes was soon profoundly felt. Gradually it was conceded that in chronic inflammations of the cecum, even more than in the acute form, internal remedies were ineffectual. Convinced by surgical achievements, although at first incredulous, internal medicine retreated from its original position.

But this complete change of view furnished to internal medicine new problems to be solved. If perityphlitis is not to become a mere byplay in the operative technic of many surgical enthusiasts, as to-day appears to be the case, although only in isolated instances, internal medicine must closely unite with surgery in weighing the indications and contraindications of operative and internal treatment, and thus satisfactorily explain their many contrasts. Moreover, it is the duty of internal clini-tions disappear or a small intramuscular abscess forms which ruptures into the peritoneum.

During this perforation the mucosa is still intact. If a fecal calculus is accidentally present in this area, it occasionally vaults the still intact mucous membrane below it. The inflammation extends more deeply into the mucosa. Diphtheritic coating and softening of the mucosa appear. Through the ruptured mucosa the fecal calculus finds its way. These fecal calculi in themselves are benign. Only when the vermiform appendix is simultaneously affected does the stone assist the process by localizing the inflammation, thus promoting a severe course and retarding the process of healing.

The estimation of anatomical and histological changes in chronic relapsing inflammation of the appendix is much more difficult.

Long ago v. Renvers and Finkelstein pointed out that anatomical an histological changes in the appendix are found at the necropsy of indi viduals who, *intra vitam*, never showed any disturbance of this orga In a remarkably thorough study Tuffier and Jeanne[1] lately determined th numerous abnormalities of the vermiform process under normal conditio in regard to size, position, and form. I think it advisable to enumera some of the most important findings of this investigation.

The position of the normal vermiform process varies from 2½ to cm. and more; it is usually from 7 to 11 cm. in length. From its inse tion at the apex the diameter is generally uniform. If the vermifor process is incised longitudinally, and the breadth of the organ is me ured, the width is found to be from 4 to 17 mm.; upon the average it from 10 to 13 mm. Yet there are many normal cases in which t organ is club-shaped, i. e., the diameter increases from its insertion to free end so that the breadth may be from 3 to 6 mm.; more frequen the inverse is found, and the caliber then gradually decreases toward free end, perhaps to such an extent that the last 2 or 3 cm. of the or are thready and resemble thin silk, the passage finally being reduced almost nothing.

The opening of the vermiform process into the cecum varies in dif ent individuals; it is circular and funnel-shaped.

The position of the vermiform appendix is sometimes

below and parallel to the ileum = **sub-ileal;**
anterior to the ileum = **pre-ileal;**
posterior to the ileum = **retro-ileal;**
below the cecum = **sub-cecal;**
posterior to the cecum = **retro-cecal;**

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[1] *Tuffier* and *Jeanne*, "Étude anatomique sur l'Appendice et la région iléoc basée sur 180 necropsiees." *Revue de gynécologie*, 1899, p. 235, quoted fro *Landau*, "Wurmfortsatzentzündung und Frauenleiden." Berlin, 1904, p. 4 u. f.

at the anterior surface covered by the cecum = internal retro-cecal;
anterior to the cecum = pre-cecal;
posterior to the ascending colon = retro-cecal;
beside the colon = para-cecal.

Sometimes the vermiform process is found in the pelvis, sometimes above it.

As to the form of the vermiform process it is usually rectilinear; often, however, it is slightly curved or bent, sometimes sharply kinked to form a right angle, or it may be hook-shaped or semilunar.

Its relation to neighboring organs also varies greatly, and naturally depends upon the manner of insertion but also to some extent upon the movable cecum and the position of the vermiform process itself. It may extend to the liver, or may be anterior to a displaced kidney; sometimes it may reach to the rectum or the sigmoid flexure, at other times nearly to the bladder.

The contents of the normal appendix differ both in quantity and quality; often a fluid or soft, doughy mass is found, sometimes small, hard, fecal particles, or it may be empty.

In regard to the so-called Gerlach valve, the recent interesting investigations of v. Hansemann[1] show that its power of closure bears an intimate relation to the presence or absence of fecal concrements. The same is to a certain extent true of regurgitation into the cecum.

The anatomical changes in chronic relapsing perityphlitis are manifold, and, corresponding to the nature of the case, they are relatively slight. According to Federmann,[2] whose description we shall follow, the mucous membrane is more or less thickened. In the mucosa we find a plentiful accumulation of round cells or connective tissue proliferation with swelling, bulging and polypoid excrescences (appendicitis polyposa). In other cases the decrease of the glandular tissue leads to atrophy or may cause partial or total obliteration of the lumen.

In other instances the appendix is more or less thickened, rigid, or erect, as Talamon expresses it, often abnormally adherent or firmly agglutinated to its surroundings. Frequently, however, it is absolutely free. The lumen of the rigid structure gapes, and is filled with a smeary, mucoid, or hemorrhagico-purulent mass admixed with portions of feces or fecal concrements of disagreeable odor. The mucous membrane is thickened, moderately tumid, injected, and in many cases is covered with ulcers. The muscular layer is distributed partly by inflammatory infiltration, partly by true labor hypertrophy. The surroundings of the process are also implicated. The appendix is more or less adherent to the cecum, to the neighboring intestinal coils, and to the parietal portion of the peritoneum, and this leads to stasis of secretion, and the accumulation of mucus and fecal constituents.

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[1] *v. Hansemann, Mittheilungen aus den Grenzgebieten der inneren Medicin und Chirurgie,* XII, p. 514, u. f.

[2] *Federmann,* in Sonnenburg, "Pathologie und Therapie der Perityphlitis." 5. Aufl., Leipzig, 1905, p. 48.

If obstruction occurs during a time in which the cavity of the vermiform process is empty, and if no virulent microbes are present in the obstructed area, inflammatory processes do not arise but the cavity becomes more or less distended, and so-called dropsy of the vermiform process sets in. The glandular apparatus undergoes a gradual retrogressive change, the epithelium is desquamated, the mucous membrane loses its original character, and all of its constituents undergo hypertrophy. The extent of the dropsy may attain the size of a fist. If during the time in which adhesions are forming many virulent bacteria are present, empyema of the vermiform process results, or a dropsy empyema may subsequently arise from secondary infection.

THE CLINICAL PICTURE OF CHRONIC PERITYPHLITIS

I. RESIDUAL PERITYPHLITIS

We have already stated that every acute attack of perityphlitis leaves traces in the vermiform appendix or in its surroundings. Nevertheless in a clinical sense the affection may be regarded as having run its course provided there are no subsequent attacks or other sequelæ. Such a cure, however, does not occur in the majority of cases, and we must always count upon the possibility and even likelihood of a relapse soon or late. Daily experience teaches us that the greater the lapse of time after the first attack the less the danger of a relapse. This, however, is by no means absolutely true, for under some circumstances relapses more or less severe may develop years after the first attack. Some authors incline to the opinion that this is not a true relapse but a new attack; yet from the fact that small pus foci may remain for years encapsulated in the surroundings of the appendix as well as that numerous mechanical changes in the appendix (obliteration, inflammatory thickening, adhesion, exudates, dropsy, empyema) follow an acute attack, the view of an acute relapse seems more likely.

A relapse following an acute attack may develop in three different ways, as follows:

1. Acute attacks occur after certain intervals and these may differ entirely from the first in severity and intensity. It must here be emphasized that a mild, primary attack may be followed by very severe and rapidly fatal, secondary relapses. To this category belongs the following case which I observed a few years ago:

S. B., a merchant, aged 24, was attacked in the spring of 1902 with mild perityphlitis and moderate fever. Against my advice the patient made a long foot tour of Switzerland in the following summer. Upon the 3rd of September, following an angina follicularis, possibly also after an error in diet, severe pain appeared in the abdomen, especially in the ileo-cecal region. The temperature rose to 101.3° F.

When I saw the patient the next day with the family physician the general condition was good. The temperature was 101.1° F., the pulse 92 and of good tension; he had slept well during the night. Above Poupart's ligament on the right there was a moderately dull, tympanitic zone which was sensitive to pressure.

Upon the 5th of September, 1902, there was jaundice and repeated vomiting occurred, *the vomitus being distinctly hemorrhagic* (guaiac test positive). Slight meteorism was present. The pulse was 96, the temperature 102° F.; the hepatic region was insensitive to pressure, and the same condition was present in the ileocecal region as on the previous day. Considering the severity of the case operation upon the same day was advised. The surgeon was E. Hahn. Finding: Gangrenous appendix with suppuration into the surroundings. Collapse and death upon the 7th of September, 1902.

Here we were dealing with one of those quite rare cases designated by Dieulafoy [1] as *vomito negro.*

On the other hand, after an exceedingly severe case, one or several mild relapses may follow.

The assumption is obvious that relapses are the immediate result of mechanical conditions (trauma, too great bodily exertion, acute indigestion, etc.), but there are also cases in which the most scrupulous care in questioning will elicit no clue to the cause.

2. The sequelæ of an acute attack are of exceedingly chronic character. Among them are chronic exudates or indurations, adhesions about the vermiform process, empyema or dropsy, thickening or kinking of the appendix, and other similar affections of the appendix or in its surroundings.

The clinical course in this group of cases is, as a rule, the same as that of chronic relapsing perityphlitis (see below).

3. The clinical picture of chronic relapsing perityphlitis may already exist but suddenly more or less severe acute symptoms develop. These are probably to be attributed to purulent residues or ulcers upon the mucous membrane of the appendix, but even sudden perforation may be the cause. The case now to be described, which at the same time proves that even after repeated rupture through the intestine with discharge of pus recovery may occur, is an illustration of this form.

Miss L., from W., aged 22, whose parents are living and well, and who states that she has always been healthy.

Toward the end of February, 1903, she was attacked by pain in the region of the cecum with vomiting, constipation, and a temperature of 102.2° F. An abscess formed, and after ten days an incision was made from the rectum and a tampon introduced. On incision a large amount of pus was discharged. Following this the patient remained in bed for three weeks but did not recuperate; she emaciated so greatly that in the spring she was sent to Pyrmont. Here, eight days later, she was again attacked by symptoms of perityphlitis, and for the second time pus was discharged through the rectum. Notwithstanding this the pain persisted for some time, and the patient was confined to bed for eight weeks. She gradually recovered, but continued very weak. Upon September 7th, 1903, she entered a sanatorium.

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[1] *Dieulafoy, Bullet. de l'académie de médec.*, 1901, No. 6.

The patient did not complain of pain, her bowels moved regularly, and she was clinically treated merely to improve her nutrition; she was carefully watched.

Status Præsens: The patient is small, undeveloped, and emaciated, with scanty musculature and a delicate frame. No edema, no eruption, no enlargement of glands. Patella reflex increased, pupillary reflex normal. Lungs normal.

Heart: Dulness normal; the second sound markedly accentuated but pure.

Abdomen: Only a small circumscribed area midway between the navel and the spinous process of the right ilium shows slight sensitiveness to pressure. No abnormal dulness.

Urine contains neither albumin nor sugar.

Bowel movement well formed with no admixture of pus or mucus.

Weight 43.5 kilograms.

Treatment: Alcohol compresses over the region of the appendix, rest in bed, stimulating diet.

Under this treatment there was decided improvement and a marked increase in weight. The patient left her bed for the first time on the 3rd of October, 1903. Her weight was then 47 kilograms.

About half-past seven in the evening the patient suddenly complained of severe pain in the region of the cecum. The temperature was 98.2° F. Cold compresses were applied, and suppositories of codein and belladonna were given; at midnight, on account of increasing pain, 0.01 of morphin hypodermically. The temperature was normal.

October 4th, 1903. The pains had been less severe after the use of morphin, but in the morning they increased. Treatment: Morphin 0.01 subcutaneously and ice-bags. In the ileo-cecal region there was an area of resistance about the size of a plate and markedly sensitive to pressure. The note upon percussion was very dull. The temperature in the morning was 99.1° F., in the evening 100.6° F. An ice-bag was ordered, also fluid diet, and 15 drops of tincture of opium every three hours.

A consultation with Prof. Körte was held on the evening of the same day. On considering the danger of an immediate operation and the fact that the temperature was but slightly elevated, the pulse 90 and strong, the decrease of the pain and the absence of peritoneal symptoms, it was decided to postpone operation and to wait until the acute symptoms disappeared.

October 5th, 1903. In the morning the condition was somewhat better; the tem perature was 98.8° F., the pulse 108. There was still sensitiveness upon pressure especially in the region of McBurney's point, and, although to a lesser extent, to th left of the navel. The resistance in the region of the cecum was apparently som what less, the percussion note rather dull, but normal in other areas. There was n singultus, no vomiting. The treatment was continued.

Afternoon: Condition less good; pulse 120, temperature 100.2° F. Toward evenin the general condition worse, pulse 128, temperature 100.6° F. The pains in t region of the cecum and also upon the left side had become very severe. Flatus w passed twice; there was no eructation, no vomiting. On account of the danger perforation Prof. Körte operated at 10 o'clock at night, the patient being und chloroform anesthesia.

The posterior surface of the colon was covered with purulent coating, the remai ing serosa of the intestine being smooth and glistening. The vermiform process w found deep down below the colon and was elevated. A point from which pus w discharged could be recognized. The appendix was about 6 cm. in length, and greatly swollen; it was extirpated in the usual manner.

Course: Without complications.

The patient was discharged cured upon the 14th of November, 1903, weighing 50.5 kilograms.

II. CHRONIC RELAPSING PERITYPHLITIS

In contrast with residual perityphlitis, chronic relapsing perityphlitis is of much milder and more benign type. Yet it is often characterized by such an enormous number of individual points, variations and complications as to make it an extraordinarily interesting affection.

In the chronic relapsing form of perityphlitis two distinctly different varieties may be diagnosticated: First, that running its course without an acute attack, or, more correctly, acute exacerbation, and secondly, that in which the chronic process is interrupted by mild attacks now and then occurring. In very rare cases (Rosenheim[1]) of these forms perforation is said to occur suddenly.

However, it is doubtful whether in these instances we are not dealing with abortive cases of residual perityphlitis. At all events a favorable and benign course in chronic relapsing perityphlitis is so much the more frequent that practically we need not consider the previously mentioned serious eventualities.

In a study of chronic relapsing perityphlitis we meet with a number of forms whose clinical symptoms are so well characterized that their recognition is of great practical importance.

We shall begin with *latent perityphlitis*.

(a) **Latent Perityphlitis.**—Although this form is not technically the subject of professional treatment, we particularly desire to discuss it, primarily because it plays an important rôle in the prophylaxis of perityphlitis. These cases may occur either in persons who years before passed through a positive attack of perityphlitis or in those who have never shown any of its symptoms. The patients who consult us for other disturbances present absolutely no symptoms referable to the ileo-cecal region, but careful and repeated examinations reveal a very characteristic sensitiveness to pressure in this area. Among 106 cases of chronic perityphlitis that I observed a year ago I noted this latent perityphlitis in 23. It is true the differentiation of such cases is difficult for, according to the reports of Keith,[2] McBurney's point is normally sensitive to pressure, but, like Lennander (loc. cit.), I emphatically deny this.

It may be admitted that McBurney's point is tender on exaggerated pressure, but a comparison of this sensitiveness with that caused by pres-tation are absent (vomiting, meteorism, facies peritonealis, singultus). The ileo-cecal region itself is more sensitive to pressure upon the first or second day of the disease than during the interval, but under suitable treatment this tenderness upon pressure either disappears in from twenty-four to forty-eight hours or ameliorates to the degree present in the intervening period.

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[1] *Rosenheim*, *Deutsche med. Wochenschr.*, 1905, Nr. 27.

[2] *Keith*, quoted from *Lennander*, "Meine Erfahrungen über Appendicitis." *Mittheilungen aus den Grenzgebieten*, 1904, Bd. XIII, p. 326.

(c) **Atypical Cases of Chronic Relapsing Perityphlitis.**—There are numerous variations from the type above described. To enumerate and to describe these would be to include an enormous number of histories. Nevertheless, among the irregular forms there are certain pathologic groups with common and easily recognizable features, and these are the more important in practice because a failure to observe them leads to erroneous therapeutic measures.

APPENDICULAR COLIC.—Among the atypical cases colica appendicularis (colique appendiculaire, Talamon) must primarily be mentioned. Under this name Talamon was the first to describe in a publication,[1] which is even to-day a masterpiece, a form of chronic perityphlitis distinguished by the following characteristic symptom-complex: The patients, frequently without a recognizable cause, or occasionally, as they report, in consequence of an error in diet, are attacked by severe colic which extends over the *entire abdomen* (not, as is stated in some text-books, limited to the ileo-cecal region). This is extraordinarily severe and lasts from 6 to 8 hours or longer. During the attack the entire abdomen is more or less sensitive and tense, just as in the case of flatulent colic, but after the attack has passed a sensitiveness in the ileo-cecal region appears and lasts for several days—and this is the most important feature in the entire symptom-complex. This subjective tenderness which becomes noticeable on active movements and even in walking and bending over corresponds to the extreme tenderness in the region of the appendix. But the subjective sensations and the objective sign of tenderness on pressure disappear after a few days, until a renewed attack produces the same symptoms. Fever and other changes referable to inflammation are absent as a rule, in the ordinary form of colica vermicularis.

What is the cause of these peculiar attacks? Most authors quite prop erly attribute them to spasmodic contractions of the appendix which resul from the attempt of the organ to expel foreign bodies, fecal concrement or other substances which have found their way into the appendix fro the cecum.

A very interesting case of this kind from Wölfler's Clinic was d scribed by Goldbach.[2]

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[1] *Talamon*, "Appendicite et Pérityphlite." *Bibliothèque médicale, Charco Débove*, Paris, 1892.

[2] *Goldbach, Prager med. Wochenschr.*, 1898, Nr. 16.

garded as a rule with but few exceptions that when such plastic changes in the region of the appendix can be determined an acute inflammatory process has always preceded.

Only under rare circumstances are these plastic changes found in the true, chronic, relapsing form. Therefore, I do not concur in the views of H. Herz,[1] who has published an otherwise excellent article concerning the condition under discussion and who states that the exudate or the tumor is the unmistakable sign of chronic perityphlitis.

Here I wish to call attention to the fact that the possibility of demonstrating thickening, adhesions, etc., at or around the appendix, is often greatly overestimated. Such adhesive changes may be suspected in the residual processes of an acute perityphlitis. Since they occur with relative frequency they are not rarely met with at a laparotomy, but under such circumstances the diagnosis is merely an accidental one. If honest, we must admit that only a *distinctly palpable tumor* or resistance permits conclusions in regard to residual processes. In my opinion there is rarely a doubt as to whether or not resistance is present, especially in the ileocecal region; but there may possibly be a question as to its character (exudate, malignant tumor, ileo-cecal tuberculosis, or induration).

In the overwhelming majority of cases of the chronic relapsing form, we find as an objective expression of perityphlitis the presence of a *painful pressure zone in the region of McBurney's point.*

This can be easily found by drawing upon the body two imaginary lines one of which connects the anterior superior spine of the ilium upon the right side with the umbilicus, and the other outlines the external margin of the right rectus muscle. The point where these lines intersect each other coincides with McBurney's point.

The importance of McBurney's point in the diagnosis of perityphlitis will be more fully discussed in the section devoted to diagnosis. Here I shall only add that the point, or, more correctly, McBurney's zone, is nothing more than a convenient landmark.

There can be no doubt that in chronic perityphlitis McBurney's point is usually sensitive to pressure, and it would be a misrepresentation of facts if we were to ignore this painful zone; in its intimate relation to the other symptoms it is naturally a clinical aid.

When the processes in typical cases of chronic relapsing perityphlitis show acute increase, this is of limited extent. The temperature is but moderately elevated (100.4° to 101.3° F.), and upon the next or the succeeding day falls to the norm. Corresponding with the temperature the pulse remains normal as to quality, it shows no intermissions, and is only moderately rapid. Except for the increase of the ileo-cecal pain the general condition is but slightly disturbed. Signs of peritoneal irricians to fill out the numerous gaps which still appear in the teaching of acute and of chronic perityphlitis.

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[1] *H. Herz, Therap. Monatshefte,* 1905, H. 3 u. 4.

In the treatment of neither form has the final word been spoken. In acute perityphlitis especially, the revision of our present therapeutic method is all the more necessary because here surgical measures do not always avert the dangers which threaten life.

From this it is evident that in the teaching of perityphlitis there is not, as is often stated, a boundary dispute between internal medicine and surgery but, on the contrary, an honorable contest, worthy of the zeal of the most noble in both branches of medicine, to attain the same end: To preserve by the best possible means the life of the patient who is in danger.

From this point of view I shall enter upon the discussion of chronic perityphlitis, which with its frequency and obduracy so often falls within the domain of the practising physician.

Before describing the clinical picture of chronic perityphlitis, a few remarks may be in order regarding the nomenclature of the disease, particularly since, by an improper designation, two fundamentally different forms of the disease are often confounded with each other.

The Americans and the English have long differentiated between two forms of chronic appendicitis: *Relapsing* and *recurrent appendicitis*. The latter term they apply to a recurrence of an acute attack, while by relapsing appendicitis they mean a chronic form developing insidiously. In French literature these forms are divided into *appendicite à rechutes* and *appendicite à recidives* (Talamon).

I recently proposed[1] the designation *residual perityphlitis* for that form which continues after an acute attack, that is, the recurring form, and for the second form which runs a chronic course the term chronic recurrent perityphlitis.

How important is the differentiation of these forms of perityphlitis will be seen from the following:

ETIOLOGY OF CHRONIC PERITYPHLITIS

What is the origin of chronic perityphlitis?

Residual perityphlitis develops, as remarked above, upon the basis of an acute attack. It may be stated, as a law, that anatomically every acute attack leaves residua. But these need not result in functional disturbance. In some cases, however, residua may be permanent, and continuously produce more or less severe symptoms.

In the chronic relapsing form of perityphlitis the conditions are reversed. Here the preceding chronic intestinal catarrh forms the chief and most frequent etiologic factor.

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[1] *Boas*, "Ueber die Diagnose und Therapie der chronischen Perityphlitis." *Deutsche med. Wochenschr.*, 1905, Nr. 27.

These intestinal catarrhs may vary in character. They may appear as an ordinary colitis mucosa or colitis membranacea, or they may be ushered in with the symptoms of chronic catarrhal diarrhea.

Habitual constipation, spastic as well as atonic, may occasionally be the precursor of chronic perityphlitis. But in my experience actual colitis is by far its most frequent cause.

It is well known that, in a small number of cases, the intestinal canal may function in a perfectly normal manner prior to the development of chronic inflammation of the appendix.

Besides chronic intestinal catarrh, in the female the uterine adnexa upon the right side play an extraordinarily important rôle;[1] disease of these adnexa may be communicated to the vermiform process, or, *vice versa*, the appendicular process may gradually spread to the right tube and the right ovary. How often disease of the appendix and a gynecological affection may simultaneously occur is shown by the fact that among 75 gynecological cases Hermes found in no less than 40, therefore in 53.3 per cent., demonstrable changes in the appendix.

Furthermore, as has long been known of acute perityphlitis, in the chronic forms traumatic influences may be of etiologic significance, and cause so-called latent inflammation to become manifest or irritative conditions suddenly to exacerbate into acute attacks. In the chronic relapsing form not only acute and powerful but continuous and weaker mechanical irritation may be a factor.

ANATOMICAL AND HISTOLOGICAL CHANGES IN CHRONIC INFLAMMATION OF THE APPENDIX

Laparotomy has permitted us a deep insight into the changes which the diseased appendix and its surroundings undergo in acute and chronic inflammation.

According to the most recent investigations of Aschoff, the inflammatory process in *acute* cases begins in the crypts of the mucous membrane. The pathogenic microörganisms which here collect in consequence of toxic action first damage the epithelium of the mucous membrane. By this effect of the toxin, or by the subsequent entrance of bacteria, a circumscribed softening of the muscularis takes place while macroscopically no changes are visible in the serosa or in the mucosa. Either these altera-

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[1] Compare *O. Hermes*, " Ueber einige Beziehungen der Appendicitis zu Erkrankungen der weiblichen Genitalorgane." *Deutsche Zeitschr. f. Chir.*, Bd. L. Also, " Erfahrungen über Veränderungen des Wurmfortsatzes bei gynäkologischen Erkrankungen." *Ibid.*, Bd. LVIII; further, *Th. Landau*, " Wurmfortsatzentzündung und Frauenleiden." Berlin, 1904, Aug. Hirschwald.

As is obvious, the group last described bears a certain similarity to the cases designated by Ewald[1] as PERITYPHLITIS LARVATA. In this form also there are atypical gastric and intestinal symptoms of varying nature and intensity but, in contradistinction to the masked cases of Treves, in perityphlitis larvata there is an evident sensitiveness to pressure in the ileo-cecal region. Such atypical cases are by no means rare, and can be correctly diagnosticated only when, as already remarked, the area of the appendix is carefully palpated in every case of visceral disease.

In my article quoted above[2] I have called attention to another variety of atypical perityphlitis distinguished by this feature: That the patients complain little of distress in the ileo-cecal region but, on the contrary, refer their symptoms to the hepatic region, while here objective examination reveals normal conditions, but extreme sensitiveness to pressure at McBurney's point.

COMPLICATIONS OF CHRONIC PERITYPHLITIS

In the residual as well as in the relapsing forms of perityphlitis complications frequently set in. In the former they may under some circumstances so completely dominate the pathologic picture that the actual focus of the disease is masked. To this category belong the dreaded hepatic abscess and pyopneumothorax subphrenicus, secondary pleurisy and pneumonia, and the embolic processes in the lungs which develop in the cours of, or after an operation for, acute or residual perityphlitis.

This by no means exhausts the number or the varieties of the complications; were we to attempt at this point to give a comprehensiv description of this grave disease which so frequently threatens life, i would be necessary to describe the entire pathology of acute perityphliti

We shall limit ourselves to mentioning a restricted number of th complications of chronic perityphlitis.

Here two groups are alike interesting: Chronic catarrh of the col and (in women) diseases of the right-sided adnexa.

In regard to the first, catarrh of the colon in its various forms a manifestations is among the most frequent and, we may almost say, dai accompanying symptoms. Here we must discuss the question in how f colitis is the cause or the effect of chronic perityphlitis. We must ad that catarrh of the colon precedes as well as follows perityphlitis, and such a manner that the bland diet necessitated and the lack of suffici bodily exercise leads to habitual constipation and gradually to true catar

The second group of complications relates to diseases of the fem genital organs, especially of the right-sided adnexa. It is not within province to describe minutely these serious affections. Those who desire detailed information are referred to the instructive and exhaustive monograph of Th. Landau, which is a critical analysis of extraordinary merit.

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[1] *Ewald,* "Die Krankheiten des Darmes und des Bauchfells," Berlin, 1902, p. 2

[2] *Boas, Deutsche med. Wochenschr.,* 1905, Nr. 27.

According to Th. Landau, besides the true diseases of the adnexa, the ovarialgia, the retroflexioversio uteri mobilis, and the dislocated right kidney may be combined with perityphlitis, and thus mask the actual underlying affection.

Besides these most common maladies, there are many others which may accompany perityphlitis. First to be mentioned here are calcareous processes of the right kidney and of the gall-bladder, which, more frequently than we are prone to believe, cause errors in diagnosis. Furthermore, as already stated, there may be a dislocated right kidney as well as perityphlitis, and if the symptom-complex be not typical this may make a differential diagnosis either perplexing or impossible. We shall discuss these difficulties more minutely in the section devoted to diagnosis.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF CHRONIC PERITYPHLITIS

The diagnosis of chronic perityphlitis may be easy, even to the novice, or, inversely, may present to the most experienced great and occasionally even insurmountable difficulty.

Starting from the principle, which has been reiterated, that residual perityphlitis and chronic relapsing perityphlitis are two clinically different forms, we shall enunciate the fundamental laws regarding the first mentioned group.

Here, primarily, the history must be regarded as the decisive factor. The more clearly this reveals the occurrence of a prior typical attack the more readily can we decide as to the nature of the possible acute relapse. Nevertheless, experience shows that milder attacks are frequently forgotten by the patient, especially if they have occurred a long time previously. If the patient is a very young person, we frequently find that such mild attacks have not been recognized by the physician owing to the insignificant symptoms, hence in a given case an existing attack may not be regarded as a relapse but as a primary attack.

Aside from this, in all essential points the diagnosis of residual perityphlitis in its acute form is evolved as in the primary attack. Besides this, however, as mentioned before, the chronic residua must be considered. In the discrimination of these the prior history of the patient is the best guide. The occurrence of fever and of the febrile movement, the duration and severity of the attack, and possible complications are especially valuable aids to the recognition not only of the nature but also of the intensity of the process.

These are the cases also in which, following the acute attack, objective symptoms persist which can be demonstrated with more or less accuracy.

The region of McBurney's point is probably sensitive to pressure, but in its surroundings we find at the same time a certain amount of thickening which may be demonstrated even on very slight percussion. In some cases an exudate may be felt. According to Ewald[1] and Sonnenburg[2] this is said to become prominent after inflation of the colon.

Sometimes the differentiation between an exudate and a neoplasm is extremely difficult, and casuistry furnishes a number of instances in which laparotomy or the necropsy has revealed numerous fundamental errors and confusions.

Among the most serious conditions leading to mistakes are carcinoma of the cecum and ileo-cecal tuberculosis.

In fact these affections, provided positive symptoms and a clear history do not guide the physician, run their course with manifestations which closely resemble chronic perityphlitis.

In the following we will present a few principles for the differentiation of these clinical pictures the prognosis of which varies so greatly, but in which, however, we cannot deny that no differentiating factors will invariably protect us from error.

Cecal Tuberculosis.—Cecal tuberculosis usually occurs in comparative youth (from the second to the fourth decade), runs an exceedingly chronic course, and frequently, although by no means always, is accompanied by tuberculous processes in the lungs. As Obrastzow correctly points out, the cecum does not form an actual tumor, but has the appearance of a rigid, irregular, nodular infiltration. Cecal tuberculosis invariably runs its course with symptoms of stenosis which in such cases are revealed in a typical manner (well developed intestinal peristalsis, alternation between constipation and diarrhea, attacks of obstruction). Blood and pus are rarely present in the feces, but tubercle bacilli may be frequently found. Fever is rare.

Carcinoma of the Cecum.—As a rule, in carcinoma of the cecum which usually occurs late in life the tumor is sharply limited. Symptoms o stenosis may be absent, or may be present in a greater or less degree Fresh or occult blood (Boas[3]) appears in the feces in from 10 to 1 per cent. of all cases; pus is either not found or is exceedingly rar Fever may occur in carcinoma of the cecum, but only in advanced stage of the affection.

Where there is no thickening, tumor formation, or even symptom c stenosis, the only objective sign is sensitiveness to pressure in the ile cecal region.

In rare cases, to find at the autopsy an inflammatory or even maligna tumor instead of the expected perityphlitis is an experience from which even the most skilful diagnostician is not exempt.

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[1] *Ewald*, "Die Krankheiten des Darmes und des Bauchfells," **Berlin, 1902.**

[2] *Sonnenburg*, "Pathologie und Therapie der Perityphlitis," **5 Aufl., Leipzig, 19**

[3] *Boas*, "Ueber occulte Magen- und Darmblutungen." ***Volkmann's Sammlu*** *klin. Vorträge*, 1905, Nr. 387.

In these and other doubtful cases I must call attention to a factor that aids materially in the diagnosis of residual perityphlitis: This is a regular temperature record continued for weeks, and especially the comparison of temperatures taken simultaneously in the axilla and in the rectum. A copy is here presented of a temperature curve which clearly portrays these differences.

I have been several times convinced that, under some circumstances, these more or less high elevations of temperature, and especially the simultaneous rectal and axillary temperatures, form the only guiding and determining symptom. The following case may serve as an example:

A girl, aged 4, daughter of a manufacturer from G. Following a doubtful acute perityphlitis she complained of periodic abdominal pains which lasted but a few seconds and occasionally ceased after vomiting. Localization of the pain was impossible. The objective finding was entirely negative, the region of the cecum being painless upon pressure. Regular and continuous records of the temperature showed occasional falls or elevations of brief duration. A diagnosis of residual perityphlitis was accordingly made and the child was operated upon by Prof. Körte. The operation revealed the following: In the mesentery a swollen gland which was extirpated. The appendix was about 10 cm. in length, contained semisolid feces, and in the center a mucous membrane cicatrix. The cecum and ascending colon were palpated and found to be normal. Course normal; discharged cured.

While in residual perityphlitis the prior history will usually give us valuable data for the diagnosis, in the *chronic relapsing form* we must depend principally upon the clinical course. Frequently this ensures a diagnosis; but there are also cases so faintly characterized that only repeated and thorough examinations will enable us to come to a decision. Among the diagnostic factors which favor chronic perityphlitis the most prominent are the characteristic subjective symptoms: The *persistent* pains starting from the ileo-cecal region and centrifugally radiating to the thigh, to the lumbar and the hepatic regions. Among the objective symptoms, as already mentioned, there is frequently nothing but the sensitiveness at McBurney's point.

In some cases another phenomenon appears upon palpation, which at this point must be described somewhat more explicitly since it is frequently and variously referred to in literature: This is palpation of the diseased vermiform process. The American surgeon, Edebohls, deserves credit for having called attention to the possibility of palpating the diseased vermiform process, and in 1894 he made the statement that this organ could be recognized in every case of perityphlitis.

That this assumption is not borne out by the facts can to-day scarcely be doubted by physicians of experience. In cases of acute perityphlitis the swelling of the mesentery, the appearance of the exudate, etc., make the conditions for palpating the diseased appendix extremely unpropitious.

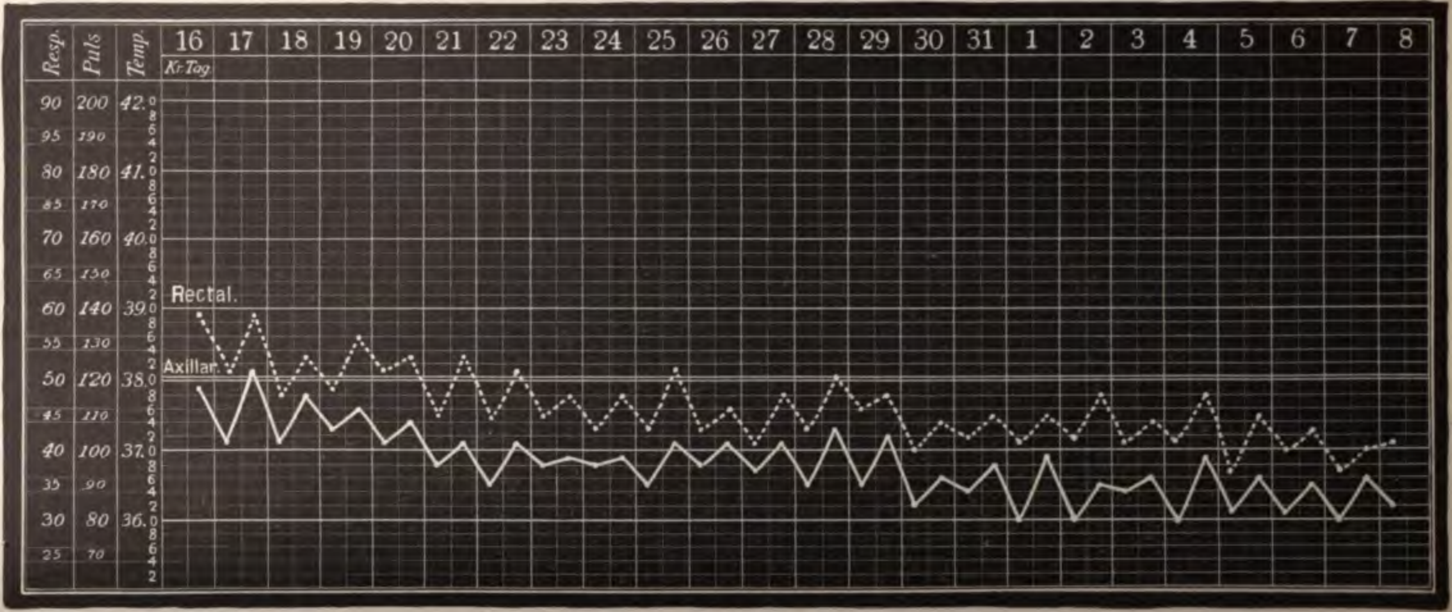

Fig. 21.—Diagnosis: Perityphlitic Exudate (relapse).

Axillary temperature: ——————   Rectal temperature: ....

The circumstances are somewhat more favorable for palpating the appendix in chronic perityphlitis, a fact to which some authors (Ewald, Hausmann) have attached a certain diagnostic significance.

In these instances we may admit that now and then when the vermiform process is in a favorable position it may be felt as a round, painful organ, sometimes with surprising distinctness. Nevertheless, dozens of cases of chronic perityphlitis may be observed in which the result of palpation is not only once but repeatedly negative. In other cases in which we think that something is palpated, the body that is felt cannot be clearly recognized, and we waver in opinion between the assumption of a contracted portion of the colon, of a coil of the ileum, of adhesive intestines, or of the rolled-up mesentery, etc.

At all events, in my experience palpation of the diseased appendix is only of diagnostic importance if we constantly find in the ileo-cecal region a distinctly palpable body which is sensitive to pressure, and which is of the same form and thickness as the vermiform process. In such isolated cases, of course, the diagnosis may be much more readily made.

The actual difficulties in the diagnosis of chronic perityphlitis depend upon our distinguishing this from other chronic forms of colitis, as well as upon the differentiation from chronic disease of the uterine adnexa.

In the great majority of cases the condition in the former is such that we must decide whether merely colitis is present or whether this coexists with chronic perityphlitis.

The differentiation is extremely difficult when the chronic colitis has its seat in the cecum, i. e., when a true chronic typhlitis exists. Then a symptom picture may develop which nowise differs from chronic perityphlitis. As a matter of fact the diseases may exist simultaneously.

The condition is somewhat more favorable when the colitis occurs in a more peripheral section of the colon, and when a severe but circumscribed pain upon pressure is produced in the ileo-cecal region. If the clinical picture of perityphlitis is simultaneously present, we can hardly doubt their coexistence.

The diagnosis is facilitated if acute, mild, febrile exacerbations appear similar to those which occur in the course of chronic relapsing perityphlitis; this impresses the stamp of certainty upon the previously doubtful case.

Nevertheless it cannot be denied that in a not inconsiderable percentage of cases doubts will arise which cannot be removed even by careful investigation and observation.

To this category belong, for example, the apparently not rare cases of so-called *pseudo-perityphlitis* to which Nothnagel[1] first called attention, and which all experienced physicians have met with.

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[1] *Nothnagel, Wien. klin. Wochenschrift,* 1899, Nr. 15; compare also *G. Singer,* "Pseudoappendicitis und Ileocöcalschmerz," Wien und Leipzig, 1905.

We are not always in a position to verify our diagnosis by operation, as was the case with Nothnagel. Nevertheless, there are some symptoms which may be looked upon as landmarks in the differential diagnosis between true and pseudo-perityphlitis. Paramount among these is the course: At certain irregular intervals the patients suffer from pain in the ileo-cecal region, but this is not associated with rises in temperature and is not increased by active or passive movements. The region of the appendix is either insensitive to pressure or the sensitiveness is only referred to the skin, and it is not circumscribed but diffuse. Occasionally cutaneous hyperesthesia alternates with normal sensation upon pressure.

I recently had an opportunity of observing a very instructive case of well-developed *typhlophobia*. In a family which was well known to me for years a fatal case of severe acute perityphlitis had occurred in a near relative. This case was for weeks the subject of conversation in the family, and one of the two daughters of the house, a girl aged fourteen, became especially concerned and uneasy. A short time afterward she complained of severe pain in the region of the cecum, lost her appetite, remained away from school, became anxious, and caused her mother a great deal of worry. The history showed that the child had never had fever. Examination of all parts of the abdomen revealed absolutely no sensitiveness nor resistance, and the adduction of the thigh which was several times attempted produced no pain. This proved that there could be no question of perityphlitis. I quieted the anxious patient, and advised her to return to school the next day and to consider herself as well. From this moment the child was well and remained so.

Besides organic and neuralgic intestinal affections, in the female the differentiation of diseases of the adnexa may come into question in a diagnostic respect. We have already indicated the possibilities which exist, and at this point attention must be called to the necessity of considering these symptomatically with chronic perityphlitis, for these genital affections may be quite analogous. In the diagnosis of inflammation of the adnexa a considerable degree of practice and experience is a prerequisite, therefore in cases at all doubtful the opinion of a gynecologist should be sought.

Besides these factors which dominate the differential diagnosis of chronic perityphlitis, there are still others which now and then come int question.

Among these are cases of calculus in the right kidney or of the *uri acid diathesis*. The regular finding of abnormal amounts of uric aci combined with small amounts of albumin in the urine, the appearance o typical attacks with their characteristic urinary changes, the demonstra tion of a gouty substratum in characteristic areas of the body, and th palpation of the kidney itself will in many cases reveal the correct diagn sis. Nevertheless, the *ensemble* of the previously mentioned symptoms not always so favorable that the underlying condition can be clearly discerned.

With reference to the kidney, its dislocation, at least so far as subjective factors are concerned, may produce symptoms which to a certain extent resemble those of chronic perityphlitis. But no actual attacks are observed, there is no typical pressure point in the ileo-cecal region, and the characteristic localization of the pains is also lacking.

Atypical cases of cholelithiasis and of cholecystitis may be confounded with chronic perityphlitis, particularly as they are not infrequently combined in the same individual. To this must be added another source of error, the not rare coexistence of cholelithiasis and colitis. An accurate history combined with cautious and repeated examinations of the liver and the region of the gall-bladder and the occasional finding of biliary coloring matter in the urine will often clear the complicated picture.

Finally, we must mention the not infrequent possibility of error from the presence of ill-defined hernia, particularly of right-sided inguinal and crural hernia. I have several times seen cases of supposedly chronic perityphlitis which were permanently cured by wearing a hernial truss.

This by no means exhausts the number of perplexities which may arise in differential diagnosis, but from what has been stated it is obvious that in some cases the diagnosis of chronic perityphlitis requires considerable acumen, practice, and experience.

One factor in particular must not be omitted from consideration, namely, that chronic perityphlitis may appear simultaneously with or independently of diseases of other organs. Sometimes we are inclined to follow an old rule and to substitute one or more pathologic varieties for each other. But nature does not sanction such a method, and we must therefore consider not only whether one or the other affection is present but—this is often the most difficult problem—whether they do not exist independently of each other.

PROGNOSIS OF CHRONIC PERITYPHLITIS

In this article we have repeatedly called attention to the fact that the prognosis in residual and in chronic perityphlitis presents fundamental differences. This is evident from the etiology of both forms of the disease. While, as is shown by the fitting designation of Sahli, in acute perityphlitis there is always "a purulent nucleus," the changes in the chronic form are referred more to the mechanical realm, and consist of kinking, thickening, adhesions and obliterations.

This is the reason why the process in acute perityphlitis may heal functionally, for example, by self-drainage (Sahli), or by rupture into are perceptible, and in how far we may prognosticate with probability the type of subsequent relapses.

Considering first the residua, the presence of an exudate may be certainly determined and its growth or retardation demonstrated by continuous observation. On the contrary, exact knowledge of the condition of the vermiform process and its immediate surroundings is much more difficult to acquire. It may still contain pus, a tendency to perforation and ulceration may still be present, the mesenteriolum may be inflamed or gangrenous, yet there may be no marked or grave symptoms to indicate the gravity of the condition. Inversely, a slowly progressive healing may take place, but whether this is permanent is a question to be answered only with the greatest reserve.

As we have already emphasized, the prognosis in a second or third attack is most perplexing. We have only to consult one of the numerous clinico-surgical reports upon appendicitis to be convinced that here every rule finds its paradigm in the inexhaustible variations of the clinical course of perityphlitis. Only ripe experience, as has often been demonstrated, warrants the following prognosis: The longer the interval after the first attack the less the danger of subsequent and severe relapses. But even here, as every one who has followed the literature of perityphlitis knows, exceptions now and then occur.

To find the proper indication for our therapeutic procedure, we must consider impartially what internal medicine has accomplished in the treatment of residual perityphlitis. There can be no doubt that the exudative form is a relatively favorable one for treatment. In such cases, indeed, local compresses of peat or salt and peat and salt baths have actually caused the exudate gradually to disappear, and thus the symptoms were either entirely relieved or diminished to a great extent.

Where there is no exudate, the result of this or any similar form of treatment is much less certain. We must be careful not to mistake a periodic amelioration of the process for recovery. As a rule, however, there is nothing to cause us to regard too optimistically an improvement in chronic perityphlitis—this is true of both forms. We have no criterion for deciding whether a process in the ileo-cecal region has healed or whether it is only latent.

For the prevention of new attacks a number of methods have been suggested. These consist mainly of bodily rest, the careful regulation of the diet, the prevention of an accumulation of feces and gas in the intestines, the long-continued use of compresses of various kinds and of different temperatures, etc. For obvious reasons it is very difficult to decide whether these measures are actually of any use in preventing relapses. Unquestionably the most important of these factors are bodily rest and proper care.

But when we regard these and similar prophylactic measures in their proper light, in the majority of cases we will cling to no illusions concerning them, even if it were possible and practicable to carry them out energetically, whereas in most cases they cannot be employed at all.

It is evident from this discussion that the operative treatment of residual perityphlitis is from principle to be preferred to any other mode of relief. Only in those cases in which we find an exudate and in which the other clinical symptoms appear favorable may we temporize. The same is true of the residua, which may exist for many years and produce no important symptoms.

After recovery from a severe attack we are placed in a position of great responsibility.

To temporize here means to allow the patient to run risks which prognostically cannot be estimated. On this point I am much more in favor of surgery than many surgeons, and I believe the future will show that I am right. Even with attacks of medium severity, I advise an operation in the interval. An expectant treatment should be instituted only in those mild cases which show but slight elevation of temperature and which terminate in recovery in a few days. Of course the individual circumstances of the patient must be most carefully considered, especially his occupation, since prolonged bodily rest is an absolute necessity.

A decidedly more favorable perspective is afforded by the chronic relapsing cases. If we bear in mind the previously stated fact that the overwhelming majority of cases of perityphlitis running a chronic course assume no life threatening aspect, we are justified in employing the remedies of internal and expectant treatment to the fullest extent.

Here also bodily rest and careful nursing are the first considerations. How long rest should be insisted upon depends, in the first place, upon the intensity of the process, and, secondly, upon the extent of time which is possible in the individual case. In the latter respect, as we have stated, our advice is disregarded more frequently than is desirable. In such cases rest for at least a part of the day or upon holidays may be insisted upon. Distinct remissions are frequently observed after several weeks of absolute rest in bed; in other cases, however, these either do not occur or are only of brief duration. And here the factor previously mentioned, the special regulation of the entire digestive function, comes into consideration.

In acute attacks there can be no doubt that indigestion plays a decidedly causative rôle; chronic indigestion also increases chronic inflammation, and *vice versa.* At this point, therefore, a few practical suggestions for the treatment of intestinal disturbances are not out of place.[1]

Primarily, as everywhere else in gastrointestinal pathology, the diet must be so regulated as to be adapted to the character of the intestinal affection. In simple, habitual constipation we institute a diet which will actively stimulate intestinal peristalsis, and one in which sugar and very sweet substances are prominent. The same result is accomplished by food containing much fat; only such articles, however, should be employed which at a low melting point are to a great extent free from fatty acids (olive oil, butter, cream, almond milk), and these constitute important aids. Organic acids, used with discretion, are of great use (buttermilk, sour milk, and the juice of apples, oranges, and lemons).

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[1] See also *Boas,* "Constipation and Hemorrhoids," in this volume.

It is much more difficult, however, in cases of chronic perityphlitis to decide the question whether food which leaves much residue and raw fruits are to be allowed or prohibited. Traditionally we are inclined to prohibit these strictly. Whether this is necessary to such an extent as is nowadays generally maintained is doubtful if we consider that an actual residue of food in the appendix is an extremely rare occurrence. In all cases the prohibition of raw fruit should extend to only those varieties which contain small seeds; vegetables, particularly those which cause fermentation, should be absolutely avoided or given only when suitably prepared. No decided objection can be raised to the use of rye bread, provided it be well baked.

The prohibition of highly carbonated drinks, of the immoderate drinking of cold fluids, and, finally, of the use of ice cream and fruit ices is quite justifiable. Here the interdiction of strong acids (vinegar), as well as of mustard, horse-radish, etc., should also be mentioned.

In case the previously mentioned diet does not regulate the bowels the most effective and least irritating treatment is by oil enemata. On principle I am opposed to actual purgatives, and only under exceptional circumstances do I advise temporarily even the mildest laxatives, such as castor oil, rhubarb, flowers of sulphur, the preparations of sagrada, and Carlsbad salt. Powerful purgatives or those which are of uncertain action are absolutely prohibited.

The treatment of habitual constipation combined with chronic intestinal catarrh is based on the same principles as that of colitis membranacea. On the other hand, the cases of catarrh of the colon running their course with habitual diarrhea must be treated by a régime the opposite of that mentioned above. Beyond a certain extent sugar and milk, perhaps eggs also, must be entirely prohibited, while, on the other hand, meat, fish, mucilaginous soups (rice, sago, barley), red wine and elderberry wine, and acorn cocoa are recommended. Fats of good quality are permissible. Here also carbonated drinks as well as all kinds of beer, ice cream and fruit ices must be avoided. In simple colitis these measures will usually be sufficient; but, if not, the well known astringents and, secondly, intestinal irrigation with astringent solutions must be considered.

Besides rest and a regulated diet, mineral spring and bath cures come into question; although they are often only palliative and transitory, there can be no doubt that these sometimes produce favorable results because individual curative factors are readily combined in this treatment, which is difficult to attain in treatment at home.

In the rare cases in which the intestinal canal is absolutely intact, we may limit ourselves to baths, salt baths, or peat baths; in others it is better to combine these with spa treatment. For the latter the waters of Carlsbad, Marienbad, Homburg, Kissingen and Wiesbaden are especially suitable. By drinking the spring water at home, or by baths, the less well-to-do classes may attempt to substitute for these cures. After the cure it is well to continue the local peat and salt compresses for a long time. The same effects may be produced by hot linseed or oatmeal poultices, as well as by compresses of absolute alcohol, after which a more or less distinct remission in the inflammatory symptoms will be observed.

Although it is undoubtedly true that these cures energetically and persistently carried out exert a favorable and palliative effect, yet the ultimate curative result is uncertain. Now and then long-continued improvement takes place and resembles a cure, but in the overwhelming majority of instances we merely attain a certain condition of latency. For some individuals this is very desirable, particularly for those who from the onset show less serious disturbances or who are in comfortable circumstances. For the remainder, however, even after the most thorough trial of this or similar cures, the result is merely armed neutrality. After long or short intervals of rest, disturbances appear, at first mild and of brief duration, but gradually becoming severe and continuous.

After considering all of the questions which here arise, we cannot refrain from expressing the opinion that the operative removal of the diseased appendix is, on principle, not only the most radical but also the most effective measure to bring final relief; of course, with certain limitations which must be carefully discussed.

First, I must reiterate that the indication for the operative removal of the appendix in chronic processes is never urgent. We may quietly await the results of thorough palliative treatment. Although, as has been stated, absolute recovery is rare, yet remissions are often very decided. Frequently we find we are dealing with appendiceal neurasthenics who torment themselves and their friends with groundless fears. Unfortunately these fears are at times augmented by some surgeons who grossly exaggerate the dangers of chronic perityphlitis. I have met a large number of these appendiceal hypochondriacs, and it proved no easy task to convince them of the mildness of their affection.

But even with more serious symptoms the indication for operative treatment is not always pressing. This is especially true in the case of aged and debilitated persons in whom any laparotomy is fraught with more or less danger. Here may be enumerated the cases with complications on the part of the heart, the kidneys, the lungs, or with diabetes and the like.

On the other hand, there are cases in which operation is undoubtedly indicated; for example, in chronic perityphlitis at the age of puberty. Even with relatively slight symptoms it is our duty in such cases to advise operation, as here the bodily rest insisted upon is directly opposed to the no less important bodily development.

The same is also true of those whose symptoms preclude their following their occupations. Here also the intensity of the symptoms must be subordinated to the fact that such persons are permanently unable to earn a living. Naturally the indication is more urgent if the symptoms become extremely aggravated and are but little or not at all ameliorated by palliative measures.

With every indication for operation it is presupposed *that the diagnosis is positive or at least extremely likely.* Errors on account of existing complications, such as intestinal catarrh or disease of the right-sided adnexa, will frequently be unavoidable; nevertheless it is invariably our duty to exhaust all of the diagnostic aids and methods at command before coming to a definite decision.

Before concluding we must discuss the dangers and results as well as the possible sequelæ of an operation. In regard to the first, statistics quite uniformly show a mortality of from one to 2 per cent. Many surgeons, however, perform hundreds of operations without a fatal case. Taken all in all we may therefore designate an operation for uncomplicated chronic perityphlitis as one relatively free from danger.

It is different with *the results.* In the first place, the influence of the operation upon the appendix and upon the remaining intestine which, as already mentioned, is frequently implicated, must be considered separately. While in the great majority of cases the effect upon the appendix is salutary, this cannot be maintained for the remaining intestinal canal. Yet we must reiterate that after removal of the appendix the cure of an intestinal affection is much more rapid because the physician need no longer take into consideration the restrictions which were previously necessary (rest, gymnastics, massage, etc.).

Nevertheless, in a few (but, as we must admit, the minority) of the cases of chronic perityphlitis, increasing experience proves that the actual curative results are by no means satisfactory.

But that I may not be suspected of disparaging the great successes o surgeons in this realm, I must call attention to a recent communicatio from one of the most distinguished operators for perityphlitis, namely the English surgeon, Treves.[1]

Among 231 cases operated upon in the stage of latency, in 45 recover was incomplete. In these 45 cases ventral hernia and all of the disturbances due to healing of the wound are excluded.

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[1] *Treves,* "The Prospects and Vicissitudes of Appendicitis After Operation *British Medical Journ.,* 1905, March 4.

Among the causes of failure were incomplete removal of the appendix (2 cases), disturbances on the part of the ovaries (9 cases), chronic or relapsing colitis (8 cases), continuance of local pain (7 cases), neurasthenia or hypochondria (5 cases), persistent pain from gall-stones (3 cases), from colic (2 cases), wandering kidney (2 cases), renal calculi (one case), soft masses in the right iliac fossa (5 cases), unknown cause (one case).

In the two cases in which the removal of the appendix was incomplete, this was removed at a second operation.

The recent reports of Haberer[1] from von Eiselberg's Clinic in Vienna are similar. Among 96 cases of operation during the interval, all of whom survived, in only 50 were the symptoms relieved by the operation. *"Forty patients, however, continued to have more or less marked disturbances, in some stubborn constipation, in some severe pain, even attacks of colic, such as they had suffered from prior to the operation."*

Reports of relapses after operation for perityphlitis are increasing in literature.[2] One of the first authors to report these occurrences was Gerhardt.[3]

I, too, have observed 5 cases of post-operative typhlitis, some of which were very obstinate. I shall quote one case, that of a colleague:

Dr. H., of F., aged 38, suffered for many years from constipation. In 1901 he had an acute attack of perityphlitis with fever and subsequently, at intervals of four weeks, five attacks, all running their course with slight fever. He was operated upon in March, 1902 (Professor v. Czerny, Heidelberg).

The conditions found were as follows: The appendix was bent, twisted in the form of a spiral, adherent posteriorly and laterally, but there was no suppuration. Following this he continued well until October, 1902. At that time there was again an acute attack of typhlitis with no rise of temperature but with great pain over McBurney's point. He was in bed for three months. Subsequently, in March, 1903, another operation was performed and the ascending colon was found to be adherent to the peritoneum. Since that time there have been no attacks, but there is still pain over McBurney's point even upon moderate movement. Moreover the patient still suffers from constipation. During an evacuation, and even for hours afterward, he invariably has great pain in the region of the cecum. The patient reports that since the two operations he is decidedly worse than before.

Examination of the cecal region reveals decided sensitiveness to pressure, particularly in the center of the cicatrix, from the operation; but even the remaining cecal region is distinctly painful upon pressure.

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[1] *Haberer*, "Beitrag zur Appendixfrage, mit besonderer Berücksichtigung von Dauerresultaten." *Arch. f. klin. Chirurgie*, Bd. LXXVI, H. 1 u. 2.

[2] *Leop. Fischl*, "Typhlitis nach Amputation des Wurmfortsatzes." *Prager med. Wochenschr.*, 1904, Nr. 7.

[3] *Gerhardt*, "Perityphlitis mit Rückfällen." *Mittheilungen aus den Grenzgebieten*, 1896, I, p. 354.

syringe, or, still better, an instrument invented by P. Cohnheim—a medium thick glass tube which is inserted within the anus and which, at the end that is introduced, is closed in front; this may readily be inflated ovally. Laterally, at the distended portion, it has an oval opening. If rotated a few times in the ampulla of the rectum we can almost always secure with this small instrument as much material as is necessary for an examination.

We find on estimating the feces of nurslings that the simple and uniform composition of the food, as compared with that of adults, greatly facilitates the process.

The conclusion drawn from this is that we must first make it easy for adults to furnish a specimen of their feces for examination; secondly, in all cases where the change in the dejecta is not so great but that it can be recognized with every kind of nourishment, we must order the patient a definite diet of uniform material. It is evident that deviations from the normal will thus be more readily recognized. With a mixed and constantly varying diet, fecal examination to a certain extent resembles a comparison of two alternating factors which simultaneously influence each other; for the composition of the feces is the result on the one hand of the nature of the food, and on the other hand of the digestive power of the intestine. If we remove from this comparison the one changeable factor, the influence of varying food, and replace it by a constant one, the condition of affairs becomes simpler. In gastric pathology this has been for some time recognized in the form of the Ewald-Boas test-breakfast as well as in the Leube-Riegel test-meal.

But to return to the primary point: In a hospital, naturally, there is no difficulty in procuring the necessary specimen for an examination. But the majority of those who suffer from intestinal diseases do not go to a hospital but to the clinic for treatment. In office practice, however, we must reckon upon the patient's delicacy in regard to everything concerning the dejecta. Here we ask the patients to bring on their next visit a portion of their dejecta, naturally that which appears to them to be pathologic, enclosed in a bottle with a wide mouth, perhaps in a preserving jar with patent lid. Some patients will refuse, for this appears to them to be worse than the disease, and only when they are accustomed to examinations of the kind, or when the physician has the necessary appliances and can furnish them with a glass jar with a proper stopper will they comply. This proposition, if I am not mistaken, emanated from Raudnitz, and, like many suggestions in practical medicine, is valuable Pasteboard may be used to place the feces in the glass jar or other container.

The point just discussed is a question of external nature. Much mor important and vital is the formulation of a test diet. Based on the pr viously mentioned considerations, this was introduced into intestinal patho ogy by Schmidt and myself. At first somewhat complicated, it was gradually so simplified by considerations of a secondary nature that the difficulties connected with it seemed to disappear. We have successfully employed this method for a number of years, and have reported the results obtained by it in our monograph upon human feces.[1]

By numerous researches we have, in the main, determined the limits of what may be regarded as normal in a test diet and what belongs to the realm of pathology, and we possess a criterion for the microscopic and macroscopic investigation of the feces. Our test diet in the form last advised by Schmidt[2] is composed of the following:

TEST DIET

Morning: 0.5 of a liter of milk (or, if milk is badly borne, 0.5 of a liter of cocoa [made of 20 grams of powdered cocoa, 10 grams of sugar, 400 grams of water and 100 grams of milk]).

With this 50 grams of zwieback.

Midday: 0.5 of a liter of oatmeal gruel (made with 40 grams of oat grits, 10 grams of butter, 200 grams of milk, 300 grams of water and one egg); this mixture should be strained.

Dinner: 125 grams of scraped beef (weighed raw), broiled only slightly so that it is raw in the center, and prepared with 20 grams of butter. With this 250 grams of mashed potatoes (190 grams of potatoes, 100 grams of milk, and 10 grams of butter).

Afternoon: The same as in the morning.

Evening: The same as at midday.

This test diet contains 120 grams of albumin, 111 grams of fat, and 191 grams of carbohydrates; it corresponds to 2,234 large calories, and almost furnishes the food requirement of a person not doing corporeal labor. For the majority of patients with intestinal disease, and with these we are dealing, it is sufficient. Individual caprices of appetite may perhaps be indulged by modifying the quantity of milk without specially influencing the result of the fecal investigation. The objection has been made that the test diet, on account of the unsuitability of milk for intestinal patients, is frequently not effective, and this is undoubtedly true in some cases; but, as Schmidt and I have ascertained by numerous investigations, it applies to only a very small percentage of patients. We should not at once yield the point if the patient states that he cannot digest milk, but should resort to a few well known artifices. As one such, I must mention the avoidance of raw milk. It should be given in the food, and perhaps with various additions. According to Schmidt, 0.3 of a gram of pure salicylic acid may be well mixed with some cold milk and added to the entire daily portion of the milk (one and a half liters) and allowed to come to a boil; this is especially beneficial. If it should be necessary, cocoa may be used as a partial substitute for milk in the trial diet.

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[1] *Schmidt* und *Strasburger*, "Die Fäces des Menschen im normalen und krankhaften Zustande." 2. Aufl., Berlin, 1905.

[2] "Die Functionsprüfung des Darmes mittelst der Probekost." Wiesbaden, 1904.

The test diet might be made up differently, but we prefer to adhere to this form, since, aside from the fact that we have tested its applicability, its modification and further testing would deprive it of its principal advantage which is that after many careful investigations of cases it has furnished us a common basis. Even with patients walking about this test diet may be well employed. I usually give the patient a printed slip with the necessary instructions, and by subsequent microscopic investigation of the fecal specimen he brings with him I am convinced that the directions have been, as a rule, correctly followed. This diet is enforced for about three days, and the dejecta of the last day are investigated. If we desire to be quite certain that the feces are the result of the test diet, or, if it is desirable (and this is important) to ascertain the rapidity of the intestinal passage, the feces may be made distinctive by administering a capsule containing 0.3 of a gram of finely powdered carmine at the beginning of the test diet. This will give to the evacuations a bright red color and the powder irritates the intestine but slightly. If, as sometimes happens, the dejecta in consequence of their own peculiar color do not show this red tint distinctly, the fine carmine granules may be readily recognized under the microscope.

As in all other investigations, fecal examinations for diagnostic purposes should follow a definite plan, and in every case a number of main points be noted. At first we naturally elicit a history of the frequency, the form, and the consistence of the dejecta, their probable admixture, and any abnormal sensations during defecation. I shall not here further discuss the individual points.

Under some circumstances it is desirable to obtain an idea of the duration of the ingesta in the gastrointestinal tract, and this may be readily determined by the administration of carmine at the beginning of the test diet, the results of which the patient may observe for himself.

The order of the actual examinations should be as follows: 1. Macroscopic; 2. Microscopic; 3. Chemical; 4. Bacteriologic.

The macroscopic examination, as must be at once premised, is the most important in practice, but it should be made in a different manner, and much more thoroughly, than is usually the case. As a rule this is followed by the microscopic examination; but, according to the circumstances of the case, or as a matter of choice, the chemical test may be first made, and this should be very simple. The bacteriologic investigation, if not included with the microscopic, is usually unnecessary; it consumes time, and except in certain typical infectious diseases the labor expended upon it is, as a rule, not commensurate with the diagnostic results.

With a flat spatula of wood a portion of the dejecta is taken from the vessel and placed in a large flat glass bowl; we observe the form of the feces (in so far as this is still retained), the consistence, as well as the difference in consistence of the various parts, the color, the odor, and the reaction. The latter is tested with moistened litmus paper, and we may note that the reaction of the surface of the fecal mass may be different from that of its interior. Naturally no opinion can be formed as to the total quantity of the feces except by observation in a hospital of the total amount discharged in at least three days, and passed in a period between the administration of two carmine capsules.

Water is added to the feces in the glass bowl, the masses are disintegrated by the spatula, and if coarser particles are admixed these may be extracted with a forceps and placed in a small glass bowl containing some water. For this purpose it is best to use two bowls, one with a black, and the other with a white lining, and to work in a clear light; Kroenig's table is very convenient (when it is necessary to search for gall-stones, foreign bodies or the like that have been swallowed, all the feces must of course be broken up, and for this purpose a fecal sieve which has several times been mentioned should be employed).

A mass of about the size of a hazelnut is placed in the mortar, diluted by the gradual addition of water until it has become of a thin fluid consistence, when this is poured into a flat glass dish for further examination.

We now consider the following points, which not only apply to the macroscopic, but to the microscopic and chemical investigations:

1. The remains of food:
    - A. Residue of food, i. e., portions of food which even a healthy intestine cannot absorb:
        - (α) The bulk of this is naturally formed by vegetable constituents or more or less encrusted cellulose. In the unopened cells we not infrequently find products which might be serviceable in nutrition, but these are unabsorbed and discharged in the feces because the husks have made it impossible for the digestive juices to reach them.
        - (β) Animal remains: Firm connective tissue, elastic tissue, bone, cartilage, scales of fish, fish bones, hairs, pieces of egg-shell, scales of crustacea and the like.
    - B. True remains of food which are capable of being digested and may be acted upon by the digestive juices, but for one reason or another have not been absorbed:
        - (α) Remains of albumin, chiefly meat;
        - (β) Carbohydrates, particularly starch;
        - (γ) Fat in different forms.

2. The kinds and the amount of the products of decomposition.
3. Pathologic admixtures from the intestinal wall:
    - A. Mucus;
    - B. Blood;
    - C. Pus;
    - D. Remains of tissue.
4. Admixtures of other kinds:
    - A. Such as have formed in the body itself (stones, parasites);
    - B. Such as have entered the intestine from without (foreign bodies).

Between 1 A and 1 B of this group there is of course no sharply defined line, which is particularly true of a mixed alternating diet with a varying preparation of the individual foods, and this cannot be too strongly emphasized. In our test diet the limitation is much more distinctly drawn.

It is worthy of note that upon macroscopic investigation of normal dejecta no undigested constituents are found which exceed in size the head of a pin. All larger particles, therefore, are of semeiotic significance, and this increases with their size. The conditions are different with a mixed diet. The dejecta of perfectly healthy persons may be well formed and of normal appearance, yet they almost invariably contain the macroscopically visible residue of vegetables, especially fragments of potatoes and turnips, leaves of lettuce, long shreds of asparagus, perhaps even entire lentils, the skins of different berries, as well as their seed. This list might be enlarged at will, and only the dilution of normal feces with water and a cursory examination are necessary in order to find these substances. Diagnostic conclusions as to the function of the digestive apparatus should be arrived at only after careful consideration. It is true we may determine from the presence of such remains that mastication was insufficient and that the food was too rapidly eaten; if these are absent, the digestive organs must, at all events, be in a healthy condition.

Of course, we do not include here the extreme cases in which numerous coarse particles are excreted, especially when the feces have lost their normal consistence, and it is evident at the first glance that these remains of food are but slightly altered, nor do we include cases of lientery. There can be no doubt as to the significance of these signs. Animal food in general, even with a mixed diet, is most easily absorbed. Nevertheless it is not unusual to find shreds of tendons or of arteries, under some circumstances also fibers of meat, and, with a plentiful ingestion, portions of tough or hard unchanged meat.

It is important for the physician to have a clear conception of thes facts, since patients, who for any reason begin to observe themselves closely frequently bring to him for examination some portion of the feces whic causes them anxiety, and the physician should have a thorough understanding of such appearances, particularly if of a vegetable nature. This enhances the wisdom of the physician in the eyes of his patient, who is not only relieved in mind by a negative diagnosis, but desires to know exactly what conditions are present. In such cases it is necessary to regulate the diet of unreliable patients, and the impression is never effaced if the error in diet is revealed to the patient from the examination of the feces.

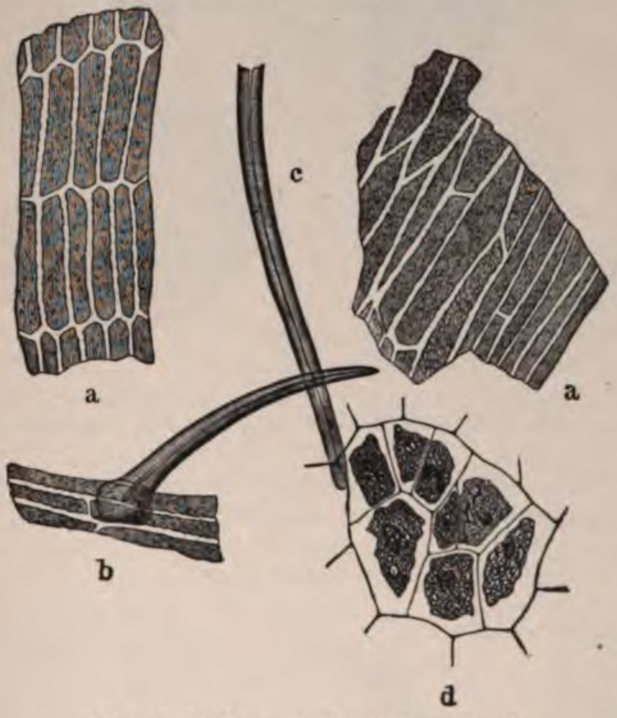

FIG. 22.—A fragment of grain from bread.

a, portions of the pericarp; *b*, portion of the pericarp with the hair; *c*, fragment of a hair; *d*, cells of the gluten layer. (Magnified 250 times.)

I shall first enumerate the food remains which most commonly appear:

While the residua of many vegetables so closely resemble each other after digestion that even the most skilful botanist cannot differentiate them, there are, on the other hand, a number of other food products, particularly from the daily diet, which can be recognized at the first glance. As a rule the aid of a microscope is necessary. A number of illustrations are given which are taken from various dejecta. They have been obtained by centrifugation of the feces rubbed up with water, and by subsequent washing of the sediment, and are much more distinct than the preparations made immediately from the feces.

We seldom fail to find in the feces the remains of grain from bread (Fig. 22). They are the more numerous and the larger, the coarser the bread which was eaten. But these characteristic constituents are also found when only gruel has been eaten: portions of the chaff and the hulls of the seed, hairs from the epidermis, and fragments of the gluten cell layer.

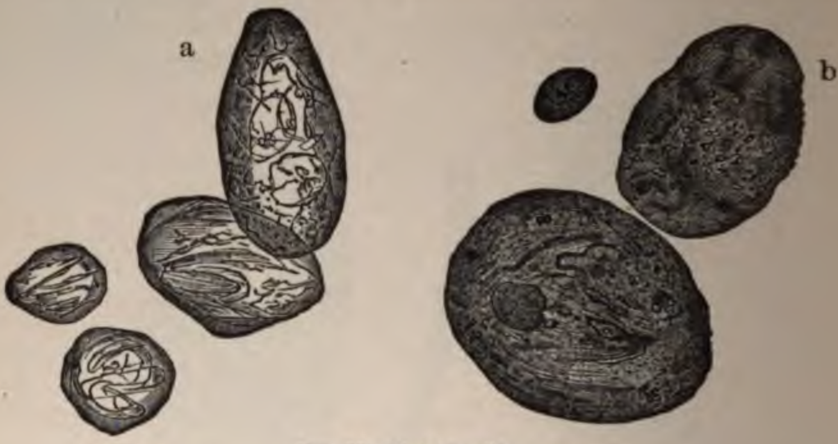

FIG. 23.—Potato cells.

a, empty, partly contracted and folded; *b*, filled with starchy material. Besides these, a starch granule retaining its external form (stained by iodin). (Magnified 250 times.)

Almost as frequently as remains of grain we find large transparent cells from the parenchyma of potatoes, and these become conspicuous on even slight enlargement (Fig. 23).

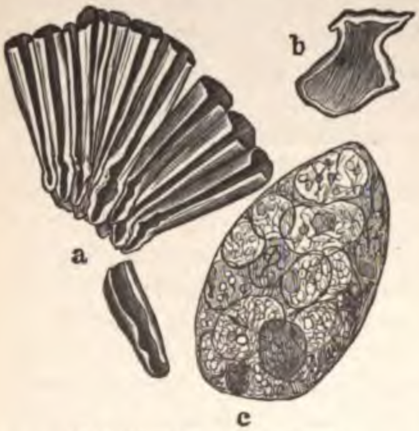

FIG. 24.—Fragment of peas.

a, palisade cells of the seed hull; *b*, a carrier cell of the seed hull; *c*, a cell from the parenchyma of the germ leaf filled with gluten and starch. (Magnified 250 times.)

These shreds are very distinct, and upon the addition of Congo red to the preparation, other delicate vegetable tissues are also revealed. As a rule they are empty and pressed flat; but some of them contain agglutinated starch, that is, dextrin, and in this case, if a sufficient quantity of Lugol's solution is employed, they change from blue to red.

The ingestion of beans, peas and lentils may be demonstrated very easily (Fig. 24).

Every preparation will reveal many glistening, greatly thickened structures in columnar form, the so-called palisade cells. Resembling these, but broader and much shorter, are the carrier cells. In peas these are detached from one another, and in the microscopic preparation they are always isolated. The carrier cells of beans, however, form a continuous layer of polygonal cells which, viewed from the surface, resemble a carpet. Each of these cells contains a crystal of calcium oxalate. The previously mentioned parts belong to the hull enclosing the seed. The large round cells resembling potatoes filled with gluten and starch granules form the parenchyma of the germ leaves.

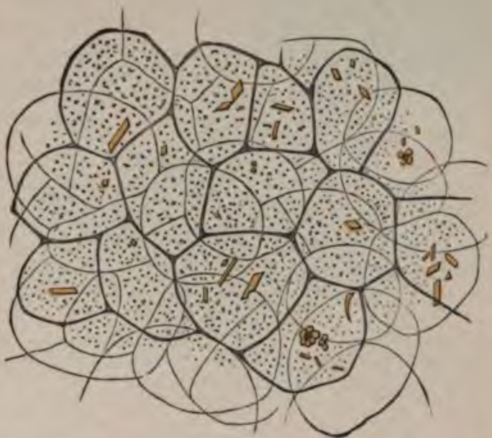

FIG. 25.—Carrot cells with crystals of carrotin. (Magnified 250 times.)

In the preparation (Fig. 25) we note very pale, large cells which contain small, isolated structures, orange to fiery red in color (carrotin), partly crystalline, partly of more irregular form; a finding which is very conspicuous and denotes the presence of particles of carrot.

The ingestion of various leaf-like vegetables is recognized chiefly from the delicate epidermis with wavy cell outlines showing intermediate spaces, the chlorophyl granules and vessels running in the ribs of the leaf serving for the conduction of water, and adorned with thickened rings and with spirals (Fig. 26).

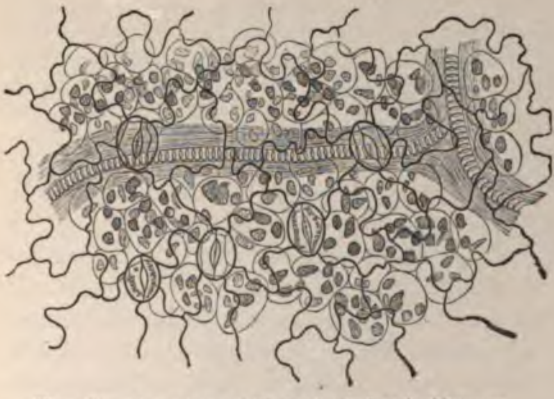

FIG. 26.—A portion of the leaf of a head of lettuce.

Above, the epidermis with wavy limited cells and with spaces; below, the chlorophyl granules in round cells and a branching bundle of vessels. (Magnified 250 times.)

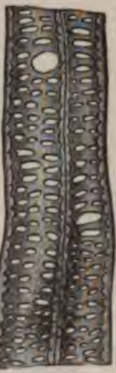

FIG. 27.—Two stippled vessels in juxtaposition. (Magnified 250 times.)

These spirals (see Fig. 27) are found less abundantly in other edible parts of vegetables, such as the stems and roots, and are less developed in the delicate leaves of flowers and fleshy fruits. They become tough and consequently so indigestible that after the consumption of even small quantities of these vegetables they invariably appear in the preparations. Often the spirals are drawn apart, or are irregularly layered among each other, or are mixed with animal fibers into a mass which cannot be separated.

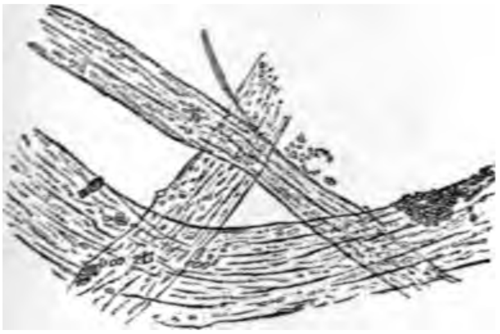

Fig. 28.—Elongated cells from the stalks of asparagus. (Magnified 250 times.)

In the next preparation (Fig. 28) we see elongated, thin-walled cells originating from a fiber of asparagus. These long threads may be observed even macroscopically in the feces; they are usually attached to shreds of tissue or the hulls of peas and beans with which they may be confounded. The microscopic picture is shown because upon superficial view there is a decided resemblance to the mucus of the feces, as may readily be seen later. Still, the more clearly outlined circumference is conspicuous. If we find the so-called raphides, parallel bundles of fine needles consisting of calcium oxalate, this is additional proof of the vegetable nature of the preparation. The characteristic cellular inclusions which are noted in intestinal mucus are, of course, lacking.

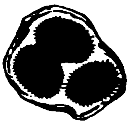

Fig. 29.

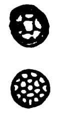

Fig. 30.

Fig. 29.—Spores of truffles in an ascus. (Magnified 250 times.)

Fig. 30.—Mildew spores of wheat (tilletia-caries), producing stone or stinking rust. (Magnified 250 times.)

The dark truffle spores which appear in the feces after partaking of this food are found in several combined transparent colorless tubes; they have fine thorns (Fig. 29) and are easily recognized. The same is true of the spores of ustilagineæ of grain (tilletia-caries) (Fig. 30) which not infrequently are found in flour. A knowledge of these is of importance, since they may appear to the novice to be the ova of intestinal parasites; for instance, of ascaris lumbricoides. Above all, they are decidedly smaller. The same error may be made with lycopodium seeds (Fig. 31) which, as is well known, are often used as a coating for pills. They appear to be partially consumed in digestion.

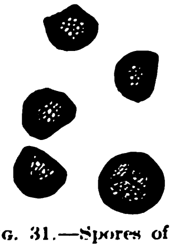

Fig. 31.—Spores of lycopodium clavatum (semen lycopodii). (Magnified 250 times.)

As the remains of fruit, I shall mention only the very characteristic core (stone cells) of pears which gives to the pulp its toughness, and which is present even in the finer varieties (Fig. 32).

The possibility of confounding the tubules of oranges with intestinal parasites must be pointed out to the investigator, especially if he limits himself to a macroscopic inspection. Virchow was the first to describe a case of this kind.

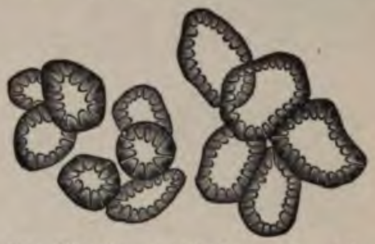

FIG. 32.—Stone cells (core) from the pulp of pears. (Magnified 250 times.)

I must devote a few words to the elastic fibers in the remains of animal food. These belong to the parts which are difficult to digest but not wholly indigestible; their dissolution may occur in the gastric juice as well as in the pancreatic juice. With a mixed diet we frequently find elastic tissue, and attach no especial diagnostic importance to its appearance. Sometimes we observe its well known double contours, beautifully wound fibers in an unchanged condition, but their continuity somewhat broken by the digestion of the intermediary tissue (Fig. 33); sometimes we see them in a half digested condition, traversed horizontally by numerous lines so that they may be confounded with fibers of wool.

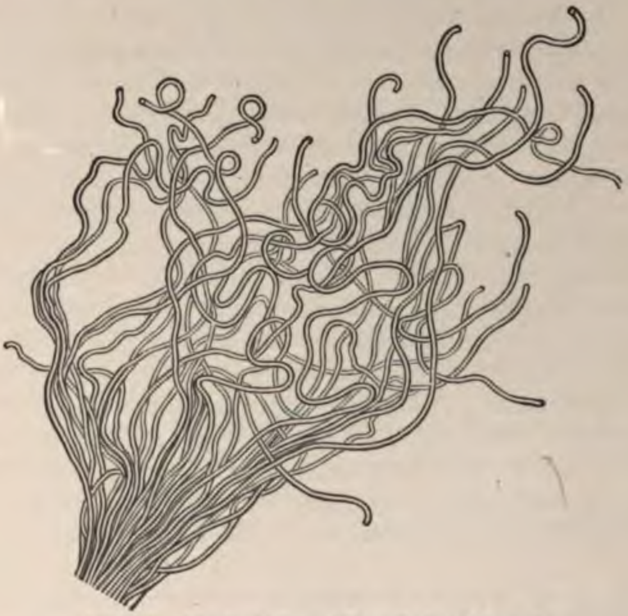

FIG. 33.—Elastic fibers. (Magnified 250 times.)

A middle position between the residua of food and the true food stuffs may be assigned to connective tissue (Fig. 34). The form in which it is ingested, more than the absolute quantity, accounts to a great extent for its reappearance in the feces. According to Schmidt the greatest bulk of connective tissue is left by smoked meat, next by raw meat, still less by fried meat, and least by tender boiled meat. Consequently, especially after the ingestion of raw ham, we not infrequently find enormous amounts of connective tissue convolutions which permeate the feces like a fine film, and which appear only upon breaking up and washing the dejecta with water.

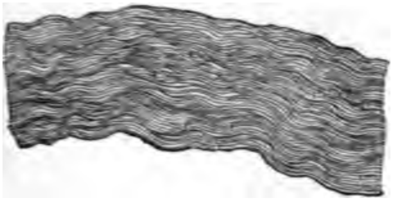

FIG. 34.—Connective tissue. (Magnified 250 times.)

The knowledge of this fact warns us to be less liberal in allowing patients raw ham than is the case at present.

We see that with a freely chosen diet the presence of connective tissue in the feces as a rule permits no conclusion as to the function of the digestive apparatus, provided large quantities have not been passed. The circumstances are different with our test diet, and upon this foundation Schmidt formulated a very valuable clinical test. Raw connective tissue, for instance, is digested only by the gastric juice, not by the pancreatic juice. Now the test diet contains 125 grams of raw scraped beef which, in order to make it palatable for those patients who refuse to eat raw meat, is broiled very lightly on the outside. Experience proves that the connective tissue in this so-called "German beefsteak" is under normal circumstances so completely digested that macroscopically no residue can be found in the feces.

Even the smallest portions visible to the naked eye point to a disturbance of digestion which, as is obvious from the preceding, has its seat in the stomach, since only this organ has the power to digest raw connectiv tissue. As a rule, in such cases there is a decrease in the secretio of hydrochloric acid. Under some circumstances, however, other disturbances of the gastric function are at fault. Thus the investigatio of the dejecta may in certain cases point to an unsuspected gastri affection, and under others may give us a clue to the malady even whe the patients refuse the introduction of the gastric tube for diagnosti purposes.

We shall now consider the manner in which the true remains of foo appear in the feces, these being chiefly represented by muscle fiber, starc and fat, the latter in varying chemical combinations. From the shape ar quantity we endeavor to form an opinion as to the function of the digesti apparatus, especially of the intestines and the large contiguous glands, t pancreas and the liver. We can then decide in how far a freely chos diet is permissible, and in how far we may succeed with the test diet proposed by Schmidt and myself.

As a rule remains of meat, even with a mixed diet, cannot be found by microscopic inspection of the feces. Therefore, the presence of large portions must be regarded as pathologic. But in the test diet the limits are more sharply drawn. Here the presence of the smallest portion of muscle, detected without magnifying, denotes disturbance either on the part of the small intestine or of the pancreas, while an affection which implicates only the stomach or the colon does not cause the excretion of macroscopically recognizable remains of meat. We may therefore to some extent form an opinion as to the seat of the disturbance of function. It is true this gives us no deep insight into the *nature* of the affection. The small intestine and the pancreas have in common, as is well known, three important functions, secretion, absorption, and motility. It would be a great advantage if we could recognize in the individual case which of these factors has suffered. As a rule, however, it is impossible to form any opinion in regard to this. A decrease in the digestion of meat, taken by itself, is no certain guide.

If the excreted fragments of muscle are found permeated by connective tissue it is obvious, from what has been previously stated, that a faulty function of the stomach is present besides the intestinal disturbance.

In the microscopic discrimination of muscle fibers we must be much more cautious than in the macroscopic recognition of residue of meat in the excretion. In man, after the ingestion of meat, we invariably find small fragments of muscular fiber in the feces, and according to the stage of digestion in which they are found three varieties may be differentiated: 1. Large, distinctly transverse, striated shreds with sharp, angular contours; 2. Medium sized particles with rounded edges, their transverse striæ less distinct; therefore a well-developed longitudinal striation is usually visible; 3. Small polygonal or round cells whose striation has been somewhat obliterated or is entirely lacking (Fig. 35).

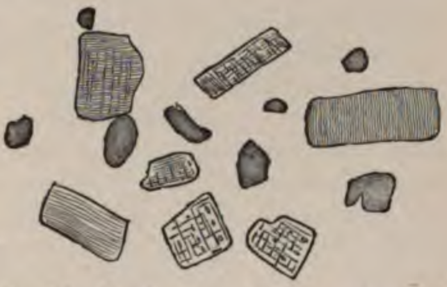
FIG. 35.—Muscle fibers in various stages of digestion. (Magnified 250 times.)

Between these three forms there are, of course, many transitional stages. After administering a test diet we may assume an intestinal disturbance if many remains of muscle of the first mentioned category are conspicuous in the preparation. In such instances, as a rule, fibers of meat may be recognized macroscopically.

From the foregoing it is obvious that a more accurate method of estimating the amount of meat digested is a practical necessity to enable us to form a definite opinion regarding this function of the intestine. The first improvement in this direction we owe to Kermauner. But his method, based upon counting the number of muscle fibers in a microscopic preparation, is laborious and does not always ensure accuracy. The method elaborated by Schmidt is better. In this the amount of the cleansed sediment of the feces is determined volumetrically, after which it is again subjected to digestion. By this process all the residue of meat which is at all digestible is acted upon by the digestive juices and dissolved. If the remaining volume of sediment is then deducted from that first found, we have the actual amount of proteids which are insusceptible to intestinal digestion. In employing the test diet, as Schorlemmer ascertained under Schmidt's direction, the difference between the normal and pathologic intestinal functions may very easily be determined. This may be utilized in scientific researches, but for clinical practice it is much too laborious to find wide application.

Until very recently, the question has been much discussed whether, and in what form, *starch* normally appears in the feces. In order to form an opinion, we must first of all separate the starch granules or agglutinated shreds which are enclosed in cells from the free starch. With a mixed diet the first forms are invariably found in the sediment of the feces. Nevertheless Nothnagel's opinion that *perfectly retained free starch granules* are normally absent is correct. Naturally we must be careful to see that starch is not liberated during the preparation by pressure upon the cells; for this reason, in the question under discussion, weight is attached only to the opinions of those authors whose researches into the dejecta have been characterized by great precision and the consideration of this point. The reports of Ledden-Hulsebosch, whose investigations were marked by extraordinary attention to detail, are especially reliable. Ledden-Hulsebosch found that even if well retained granules do not appear with a mixed diet, nevertheless agglutinated starch in the form of layers and shreds lying free is detected. Naturally this can only be determined by the careful investigation of the cleansed sediment of the feces. The addition of iodin to the simple microscopic preparation, even if a large amount is added, frequently produces no result, although it can be certainly proven by other methods that starch is present. This certainty is attained if we subject the feces to a "fermentation test," or, as Schmidt generally expresses it, to an "incubation oven test"; i. e., the feces diluted with water in a suitable vessel are subjected to the heat of an incubation oven after which we note whether processes of decomposition appear and also their action. If the absorption of food has been so complete that the feces show in the main only a residue, no decided change takes place after about twenty-four hours in the incubation oven. If, however, constituents of the food which may be readily acted upon are admixed (and this is particularly true of albumin, starch, and to a less degree of fat), these are broken up by the microörganisms of the feces on the formation of the characteristic products of decomposition. According to the character of this process, we differentiate between fermentation and decomposition.

Fermentation, as a rule, runs a more rapid course than decomposition, and is attended by the production of odorless gas and an increase in the acidity of the feces, the color of the dejecta becoming lighter. The process of fermentation depends upon the decomposition of carbohydrates, particularly of starch, which is changed into sugar by the diastatic ferment always present in the feces, and is further disintegrated by the bacteria present in large amounts. *Decomposition* is slower; gas, as a rule, is developed in less amount but is characterized by its disagreeable odor. In this process the feces become alkaline, their color darker. This is the consequence of decomposition of albumin, and less of the remains of food which do not so readily undergo decomposition, as, for instance, the pathologic products which are excreted from the intestinal wall, such as mucus, pus and blood.

A positive reaction of the fermentation test, therefore, shows us the presence of carbohydrates in the feces. A point to be observed is whether they are of such nature that they may be readily acted upon by the bacteria, and are therefore susceptible to intestinal digestion and, with sufficient power of the intestine, would have been digested. We are dealing with actual *food residue,* not the remains of food. In accordance with the fact previously mentioned that we frequently find free remains of starch, the fermentation test with a mixed diet is often positive. It is a fine reagent, and gives results even in cases in which the ordinary examination, the usual microscopic investigation of starch, is unsatisfactory. If the amount of carbohydrates in the food is limited to a certain extent, as in our test diet, fermentation will normally not be present, as the intestine is fully capable of absorbing the amount of carbohydrates it contains. In some cases, however, even after the test diet, the feces contain starch and ferments; we are then justified in speaking of the "insufficiency of starch digestion."

At the same time, the other foods may be well absorbed. Such an occurrence is not rare, and it forms a symptom-complex which Schmidt and I have described as "intestinal fermentative dyspepsia." In this condition, so far as our observations extend, the stomach, colon, and probably also the pancreas, function in a normal manner; since the labor of starch digestion is laid principally on the small intestine, we have reason to believe that we are dealing with a disturbance of the small intestine, probably with an anomaly in the secretion of the intestinal juice, a secretion which has normally a powerful amylolytic action. To demonstrate fermentative dyspepsia while employing the test diet, we use the fermentation test, a method which, as we learn by experience, is superior to searching the microscopic preparation for starch.

I shall demonstrate from the feces of a patient the relation to intestinal fermentative dyspepsia. These dejecta were from a powerfully built, fairly well nourished man in middle life, who for months had complained frequently of abdominal pain particularly in the region of the navel, also of a certain lassitude and malaise. There were no gastric symptoms. From time to time there was constipation; frequently, however, there were two or three thin, pappy stools daily. Actual diarrhea was never present. Upon careful questioning the patient recalled that the dejecta were sometimes foamy, of a light color, the odor sometimes resembling that of old cheese. On such days the fecal evacuations occurred three or four times in twenty-four hours. The consistence, however, was not fluid but always pappy. External examination of the chest and abdominal organs revealed only a moderate distention of the abdomen and a diffused sensitiveness to pressure around the navel, particularly to the left. The epigastrium was not painful upon pressure; the tongue was slightly coated. An examination of the evacuated gastric contents showed them to be normal. The feces were then examined, as it was to these the history pointed.

The patient was instructed to bring a specimen of the dejecta for examination, but I shall not relate what was then found as the investigation revealed nothing definite. The man was then placed upon our test diet. On inspecting the feces it was noted that the consistency was pappy, somewhat thinner than normal, the reaction moderately acid. Otherwise both microscopically and macroscopically no abnormal admixture was found. Meat and fat were well absorbed. On the other hand, the number of potato cells was conspicuous; most of them were empty; with iodin they occasionally revealed demonstrable starch. No free remains of starch were found. The fermentation test was then made in the following manner:

About 5 grams of the well mixed feces were disintegrated by means of a suitable instrument (a wooden spatula). If the feces are hard less should be taken, if thin a correspondingly larger amount, so that approximately about the same amount of dry substance is worked up. The fermentation vessel[1] (Fig. 36) is taken apart so that only three rubber plugs are combined with one another. In *a*, the lower chamber of the fermentation tube, the feces are stirred up with water, after which the rubber plug is placed within the tube to prevent the formation of air bubbles. The chamber, *b*, is filled with water and also closed with a smaller rubber plug, so that no air bubbles can form in the tube. Finally, the tube, *c* which has at its apex a small hole for the escape of air, is placed in position. The illustration shows how far the glass tubes which are united project into the lumen of the larger tube. After the parts of the apparatus are attached, it is placed for twenty-four hours in an incubation oven heated to 37° C., or into a water bath of the same temperature. If gas develops from the feces, a corresponding amount of water is forced into the ascending tube, *c*, where the height of the water may be read off. The reaction of the feces should be tested with litmus paper before and after fermentation. The diagnostic employment of fermentation is only indicated after the test diet recommended by Schmidt and Strasburger.[1]

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[1] Made by C. Gerhardt in Bonn.

Of course, any other fermentation apparatus may be employed; for instance, those used in the examination of urine. Our fermentation tubes, however, permit a more accurate opinion as to the amount of gas formation since nothing is lost in fermentation, and the amount of displaced water may be more easily read than that of the gas which has been formed, since this in part permeates the feces in the form of bubbles.

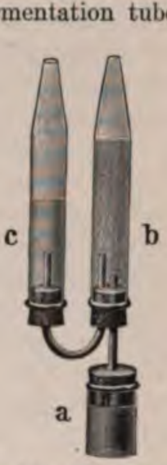

FIG. 36.—Fermentation tubes with contents.

Positive reaction of the fermentation test. The quantity displaced in tube, *c*, is greater than that of the gas in the upper part of tube, *b*, as a portion of the gas has been retained in the feces in the vessel, *a*. (One-fifth of natural size.)

The test is to be looked upon as positive when at least a third of the tube, *c*, is filled with displaced water. This happened in our case. The examination with litmus paper showed a positive acid reaction of the fermenting feces, and we then determined that the gases formed were odorless. In the patient in question we could scarcely have formed an opinion as to the malady present except by this test. It is true our diagnosis is a purely functional one, the anatomical disturbance causing it being still unrecognizable. But in the great majority of cases it is probably due to catarrh of the mucous membrane of the small intestine.

By the aid of the fermentation test we have come to a conclusion as to the nature and the degree of the processes of decomposition to which the feces, if we may be permitted the expression, are predisposed. In well developed cases this decomposition attains its full development in the intestine itself, and the dejecta when discharged are either light, foamy, and acid, the odor reminding us of butyric acid, or they are dark, have an alkaline reaction and a fetid odor, which, under some circumstances, may be even putrid. In comparison with the preceding, the disturbances last enumerated are always more serious, and correspond with what we learn from the subjective examination of the patient.

I have included in my discussion of the absorption of albumin and positive, and it is quite likely that this accounted for the peculiar color of the feces. We then instituted another test, an attempt to prove the presence of biliary coloring matter. For this purpose I mixed a large portion of the feces with dilute acid, heated it to split the soaps, and after cooling I added ether. Thus it was possible to remove a portion of the fat, after which the color of the remaining feces became a distinct brown. I subsequently attempted to prove the presence of biliary coloring matter, and for this purpose added to the fresh feces some hydrochloric acid-alcohol. When hydrobilirubin or leukourobilin (bilirubin is not found in the normal feces of the adult) is present, the coloring matter may be obtained from the feces in the following manner: To the clear filtrate of the extract, I add a few drops of ammonia and a solution of zinc chlorid, whereupon, after some time, and in a clear light, a beautiful greenish fluorescence is noted. Leukohydrobilirubin, which is demonstrated by this test, is changed back into hydrobilirubin. Another portion of the extract I subject to a spectroscopic examination. With a suitable dilution the characteristic absorption bands are found to be very distinct in the markedly broken portion of the spectrum, between the lines b F, which move somewhat to the left when the fluid is rendered alkaline. This, therefore, furnishes proof that the biliary coloring matter has been excreted and has reached the feces. That a too great reduction of coloring matter was the cause of the white color of the feces as well as their richness in fat is improbable on account of the great amount of fat present in the stool, for the presence of leukourobilin does not normally cause a disturbance in the digestion of fat.

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[1] Compare *Schmidt* and *Strasburger*, "Die Fäces des Menschen." Berlin, Hirschwald, 2. Aufl., 1905; or, *Deutsches Archiv für klin. Medicin*, LXIX, p. 570.

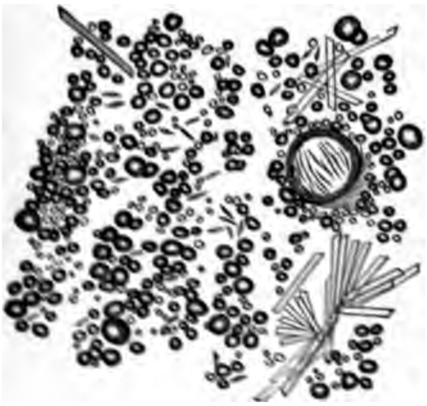

Fig. 38.—The same preparation as Fig. 37, but after heating with dilute sulphuric acid.

The fatty acid is seen in drops. In the interior and at the periphery of a large drop, fatty acid needles are beginning to crystallize. The large crystalline needles are calcium crystals and prove that the base of the soap is calcium. (Magnified 500 times.)

The whitish appearance of the stool is therefore due to the immoderat amount of fat present, and the question now arises to what the insufficien absorption of fat is to be attributed. As the absence of bile cannot b considered the cause, the possibility remains of a disturbance in the secr tion of the pancreatic juice, or a disturbance of intestinal digestio The examination of the feces also gives us many points which aid in th elucidation of this question.

Absence of the pancreatic secretion often leads to a deficient splitti of fat, which is excreted in the feces chiefly as neutral fat. Only the most minute quantitative chemical analysis will positively demonstrate this. Nevertheless if numerous soap crystals are present when we examine the microscopic preparation, or, on the other hand, neutral fat appears in the form of drops, we may form a definite opinion. Notwithstanding decreased fat splitting, the total quantity of fat excreted is not necessarily increased; but, besides a sufficient splitting of fats, a large amount of fat may be observed in the feces in disease of the pancreas. The picture therefore varies greatly.

The absence of the pancreatic secretion also causes, as a rule, a decided decrease in the absorption of albumin which may be recognized in the microscopic preparation by the presence of numerous fragments of muscle with sharp edges.

Ad. Schmidt has called attention to another significant feature. It depends upon the fact that cell nuclei are digested only by the pancreatic juice. If this secretion is absent the cell nuclei are found, for example, in the muscle fibers. Schmidt considers it advisable to give small pieces of meat which have been hardened in alcohol, and subsequently placed within very thin gauze coverings. These are easily detected in the feces, and if in natural condition, otherwise after staining, they are examined for the presence of cell nuclei. This test can be utilized only if the result is positive, since under some circumstances intestinal decomposition alone is sufficient to destroy the cell nuclei.

In doubtful cases the investigation of the feces after the administration of pankreon may give us light.

Other tests for the diagnosis of disease of the pancreas must be omitted since they do not pertain to our theme. But a point to be specially emphasized is that the symptoms of disease of the pancreas appear, as a rule, only when the gland is nearly or entirely destroyed. They may be absent even with complete occlusion of the excretory ducts, and this leads us to think that the secretion may find its way into the intestine by some other channel. Hence mild disturbances of the pancreatic function invariably escape recognition.

If, in diseases of the pancreas, the increase of fat in the dejecta is due to intestinal disturbances, we often find an increase also of the remains of meat, but in this case it appears to be less marked. As the excretion of fat in intestinal diseases is chiefly caused by deficient absorption, eventually other signs of a lack of absorption appear, and confirm the diagnosis.

The presence of dissolved albumin is especially important because this is not found in normal feces. Great care is necessitated in its demonstration, as we have learned by the experiences of the last few years. It is especially important not to confound it with the nucleo-proteid of the feces which is invariably present. The easiest and most reliable method appears to be that of O. Simon[1]: Water is poured over the fresh fece and the mixture is allowed to stand for some time, after which the liqui is carefully drained off and filtered. To make it perfectly clear diatomace ous earth is added, the mixture is shaken, and again filtered. Dilute aceti acid is then cautiously added to precipitate the nucleo-proteid, and the mix ture is again filtered. It is difficult correctly to estimate the amount of aci necessary, for if the acetic acid is in excess it will again dissolve the nucleo proteid. If, after the addition of acetic acid, a slight turbidity appear which is very difficult to filter, another acetic acid test may be made wit larger amounts, and subsequently a test for albumin with dilute potassiu ferrocyanid solution. The precipitate which appears with this must b decidedly more marked than in the first test if it is to be utilized as a proo of the presence of albumin (native proteid bodies and albumoses).

We can also attempt to demonstrate albumin in a watery extract o the feces by the aid of the biuret test (caustic potash and dilute coppe sulphate). This is, however, often difficult because the coloring matte of feces (hydrobilirubin) reacts with the biuret test just as it does wit albumin. On using the simple methods which are employed to remove th coloring matter a portion of the dissolved albumin bodies is also los they are usually present in only very small amounts; Ury's[2] proces which is the most accurate one, is much too difficult for the gener practitioner, especially as it necessitates the removal also of the nucle proteid of the feces.

Leaving the discussion of Simon's test for dissolved albumin, I mu return to the investigation of the feces under consideration, since it wa demonstrated that they contained dissolved albumin in considerable amoun The signs previously enumerated as favoring pancreatic disease were e tirely absent.

The result of our investigation therefore showed that the marked i crease of fat in the feces was due to the cessation of absorption in t intestine. The question then arises, What is the cause of this disturban in absorption? Here, however, I cannot minutely discuss this questio for the investigation detailed gave no further results, and it is my prima object to describe a number of methods of investigation and diagnos considerations which may be utilized when the feces are difficult to analy

When describing the methods of demonstrating biliary coloring matt I omitted to call attention to a test which is of general application, a in a diagnostic respect leads to important conclusions. This is the cor sive sublimate test invented by Ad. Schmidt, to which a few words w be devoted. This test shows us by a color reaction whether the mass feces or any isolated portion of the same is colored by bilirubin or by reduction product, hydrobilirubin. The former is changed to green by corrosive sublimate test, the latter to rose red. We now know that the contents of the small intestine are normally stained green by the corrosive sublimate test, those of the colon red, and that the ileo-cecal valve forms a sharp boundary. This is due to the fact that below this region the processes of decomposition set in which, hand in hand with reduction, transform all bilirubin into hydrobilirubin. Only in the nursling, in whom intestinal decomposition is normally absent or very slight, are the conditions different, and here the coloring matter of the feces wholly or in part is composed of bilirubin. If we find unaltered biliary coloring matter in feces other than those of nurslings, it is obvious that the coloring matter is from the small intestine or the upper part of the colon, and in consequence of too rapid propulsion or other less obvious reason it has permeated the feces unchanged. At all events we are dealing with a disturbance which, as a rule, may be referred to the small intestine. The examination of the feces for residue of meat, the results of the fermentation test, the characteristic mucus particles, will then serve to localize the affection. If the feces contain much bilirubin they will be of a bright, golden-yellow color, like the feces from healthy, breast-fed children. This is found in the rare cases designated by Nothnagel as jejunal diarrhea. More frequently isolated fragments, particularly of muscle fiber and mucus, show a greenish discoloration with corrosive sublimate.

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[1] *Archiv f. Verdauungskrankheiten*, 1904, IX, p. 197. [2] Ibid., Bd. IX, 1903, p.

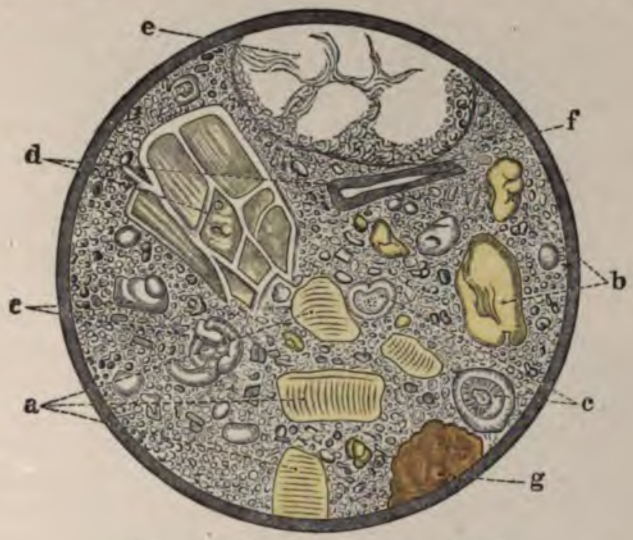

FIG. 39.—Normal feces from a test diet.

a, muscle fiber remains; *b*, yellow calcium salt; *c*, soaps; *d*, chaff remains; *e*, empty potato cell; *f*, detritus; *g*, cacao remains. (Combination picture.)

To carry out Schmidt's corrosive sublimate test a portion of recently evacuated feces, the size of a hazelnut or walnut, is carefully diluted with a considerable quantity of a concentrated bichlorid solution. This mixture is permitted to stand for several hours or a day in a broad, flat glass dish with a cover. The time consumed is, of course, an objection to this method. The green color of the familiar Gmelin reaction immediately shows the presence of bilirubin. Unfortunately, however, it cannot be used for the finer tests. If bilirubin is present only in microscopic amounts, Gmelin's test is unreliable.

Before concluding this discussion of the remains of food in the feces, I wish to call attention to two illustrations, after Schmidt, of the "test of the function of the intestine by means of the test diet." In a combination picture they exhibit the constituents of the feces after a test diet, the first illustration showing normal, the second, pathological conditions.

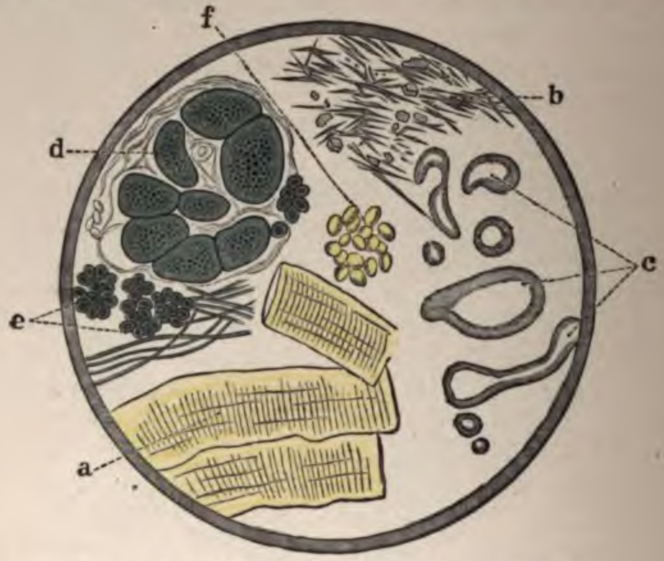

FIG. 40.—Pathologic constituents in feces after a test meal.

a, large fragments of muscle; *b*, needles of fatty acids and soaps; *c*, neutral fat; *d*, starch granules in a potato cell; *e*, granulose containing bacteria; *f*, yeast cells. (Combination picture.)

A large proportion of the total fecal mass is formed by bacteria; this is at once corroborated by the investigation of a microscopic preparation. A large portion of what is otherwise designated as detritus proves, upon close investigation, to be more or less well preserved microörganisms. By a special process I mechanically separated these from the other constituents of the feces, and by weighing ascertained that with an easily digested diet bacteria formed about one-third of the total amount of solids. Upon an average 8 grams of dried bacteria are passed during twenty-four hours. An estimation of their number based on this weight and upon the medium size of the bacterium coli communi gave the extraordinary figure of about

128 billions, a sum absolutely beyond our powers of conception. In this article I cannot enter more minutely upon the subject of fecal bacteria. So far as the familiar pathogenic agents which find ingress by way of the intestine are concerned, they have been referred to in special articles. The question of the importance of the other microörganisms in the feces is, however, much too complicated to be briefly considered. With the exception of the bacteriology of the feces of nurslings, the diagnostic conclusions reached have been comparatively few.

Nor can I devote much attention to the description of the animal parasites. (See volume on "Diseases of Metabolism and of the Blood, Animal Parasites, and Toxicology.")

In a diagnostic respect, the demonstration in the feces of certain pathologic products secreted by the intestinal wall is very significant. In point of frequency, this is especially true of mucus and of blood. The presence of pus or other tissue constituents is much more rare.

Mucus is found in the feces in varying form and distribution. Sometimes it is intimately admixed; this occurs only with a pappy or fluid consistence of the excrement, and, under some circumstances, the flocculi are then so small that they cannot be readily recognized; at other times coarse shreds which originate from the grooves in the haustra of the colon are adherent to the surface of the feces, which are frequently hard. Very dry dejecta are occasionally coated with a thin film of mucus resembling a varnish. Excrement which consists exclusively of large mucus masses, perhaps in the form of tubular casts of the intestine, is found in enteritis membranacea or in colica mucosa. Less massive quantities of hemorrhagico-purulent mucus are voided by patients with dysentery associated with marked tenesmus. The mucus in the feces is sometimes glassy and transparent, sometimes very turbid or even white, according to its impregnation with fat and its permeation with cells. Its consistency varies from that of extreme fluidity and elasticity to the toughness of thin leather. When larger amounts are evacuated confusion with animal membranes or connective tissue convolutions from the food, or even with animal parasites, is quite possible.

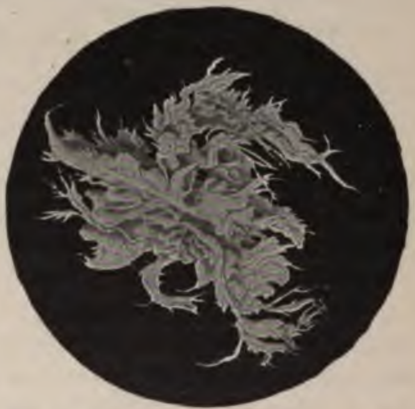
FIG. 41. — Connective tissue from the feces. (Natural size.)

The first illustration (Fig. 41) upon a black base shows a coarse shred of connective tissue detached from the feces and washed clean, and beside it a mucus shred of the same size (Fig. 42). The latter differs from the white connective tissue by its dirty color; the individual processes are not so sharply defined; they are more rounded, and in the thin portions are somewhat transparent. In doubtful cases a microscopic examination must always be resorted to, and this will at once enable us to form definite conclusions (compare Figs. 43 and 44).

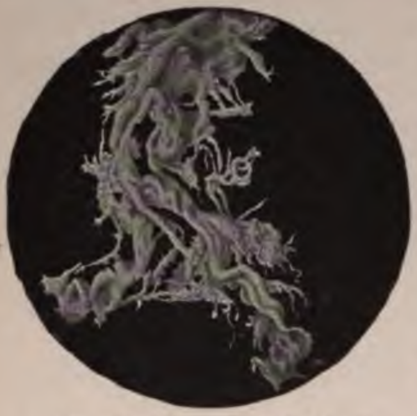

FIG. 42.—Mucus from the feces. (Natural size.)

The small, transparent, mucus flocculi which are most common are best recognized when the feces are thoroughly diluted with water and spread out in a thin layer upon a glass plate which is lighted from underneath, or in a glass vessel which is held toward the light, the feces then being permitted to trickle down the sides of the vessel. Mucus flocculi may then be noted as circumscribed transparent particles. Even with these precautions mucus may be confounded with swollen fragments of undigested vegetables or fruit (for example, potatoes, carrots, pears), and microscopical proof should always be obtained since this leads to definite conclusions.

A feature of mucus to be recognized microscopically is the delicate, often scarcely visible, contour, as well as a number of irregular lines in groups and running almost parallel (Fig. 43). After the addition of dilute acetic acid to the preparation and a thorough mixing, the precipitate of mucin causes a reaction characteristic of this tissue; namely, dark lines standing out prominently (Fig. 44). They are the expression of the never absent overlapping, and the entire preparation resembles a veil which is partially folded. Admixtures of every kind, detritus, bacteria, remains of food, above all certain cells, are found almost invariably in the mucus. Only when the mucus is very glassy are these absent; their amount is increased the more turbid the mucus and the whiter its color. For the most part they consist of so-called disintegrated intestinal epithelia, i. e., homogeneous cells the component parts of which can no longer be recog nized. This loss of structure Schmidt proved to be the result of imbi

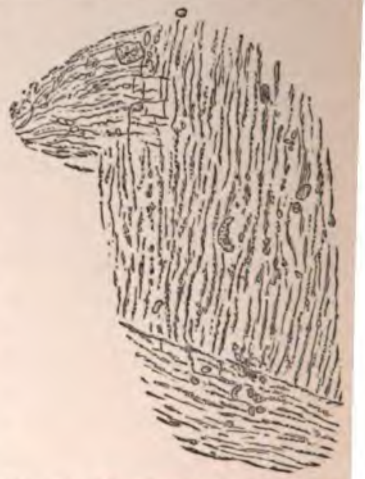

FIG. 43.—Mucus with isolated disin tegrated epithelia. (Magnifie 250 times.)

bition with soaps, for if the preparation is heated with acetic acid the cells become clear and the nuclei visible. The demonstration of half digested cells in the mucus is of significance. In such cases only cell nuclei may be found, and around these in cellular arrangement granules or crystals of bilirubin. The mucus itself is stained yellow by bilirubin. These signs certainly indicate that the mucus originates from the small intestine.

The fact that intestinal mucus contains epithelia is noteworthy when contrasted with the constituents of the mucus in sputum in which leukocytes predominate but only isolated epithelium is found. Under some circumstances, of course, pus cells also may be found in intestinal mucus. This is then of special import, a fact to which I shall revert later.

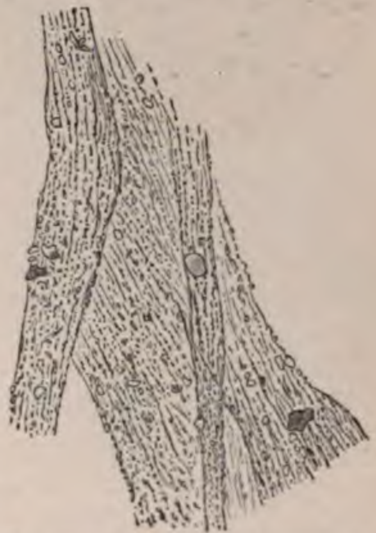

FIG. 44.—The same after thorough admixture with dilute acetic acid. (Magnified 250 times.)

The diagnostic importance of mucus is based on the fact that every admixture of mucus to the feces, macroscopically and microscopically evident, denotes a pathological condition, this condition with but few exceptions being *catarrh* of the intestine. The meconium plug of the new-born has special significance, this being a short column of mucus which is expelled by the nursling even before meconium is excreted. This meconium plug was discovered only a few years ago by H. Cramer; that it escaped observation is probably due to the fact that it was covered in the diapers. Small mucus constituents are normally found in the feces of nurslings up to about the second week. It is still a question in how far the large mucus masses of colica mucosa which are usually passed without any fecal admixture, and which are regarded by Leube and Nothnagel as the result of a neurosis of secretion rather than an enteritis membranacea, may be referred to a mild catarrh.

It may be said of the thin mucus coat which is occasionally observed upon the surface of hard fecal masses, and which resembles varnish after drying, that it is quite within the limits of physiologic conditions.

In the decision as to the *seat* of the catarrh, the question from which portion of the intestine the visible mucus present in the feces originates is of great diagnostic significance. We will observe that the excretion of mucus in the small intestine is less than that in the colon; furthermore that mucus is very readily digested, and when it is excreted in the small intestine it is subjected to the action of the proteolytic ferment, and that, therefore, by far the greatest quantity of mucus particles admixed with feces originate in the colon.

When there is highly increased peristalsis of the small intestine and of the colon, mucus finds its way from the small intestine into the feces, which then, under circumstances already referred to, contain partly digested cells stained with bilirubin. This is chiefly found in enteric fever and in severe intestinal tuberculosis. Only the smallest, most finely distributed mucus flocculi can possibly originate in the small intestine; coarser particles undoubtedly come from the colon. A more definite localization in the course of this organ is only possible if we assume that the seat of the catarrh is the higher the finer the mucus flocculi, and the more intimate their admixture with feces. We may obtain some idea of the intensity of the inflammation from the number of admixed cells. As a rule, a white appearance of the mucus in consequence of the presence of considerable cylinder epithelium of the intestine much more certainly indicates catarrh than the presence of mucus which is glassy or but slightly turbid. Although the presence of mucus in the feces is conclusive, we dare not assume from the absence of mucus that catarrh is not present. Under some circumstances mucus may be found in the colon by the test washing of the bowel as advised by Boas.

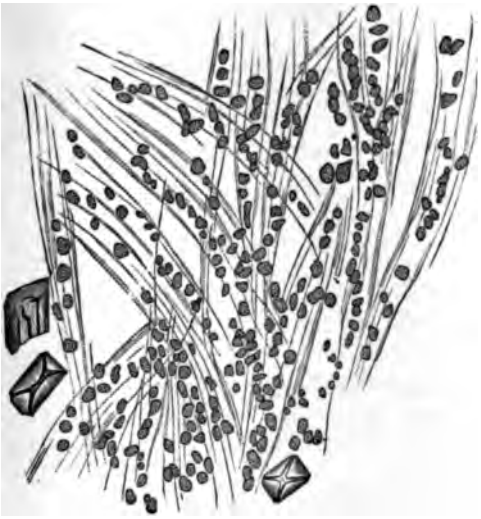
FIG. 45.—Mucus with numerous red blood-corpuscles in the process of destruction; with these are crystals of triple phosphates. (Magnified 250 times.)

Small amounts of pure *blood* upon the surface of feces are found especially in the case of hemorrhoids, perhaps mixed with tough mucus. The muco-purulent dejecta in intestinal invagination are thin and profuse, and the so-called muco-purulent *lotio carnea* of dysentery also are often very profuse. The erythrocytes are readily destroyed in the intestinal mucus, hence they cannot be demonstrated by the microscope. In Fig. 45 we see red blood-corpuscles of a rusty appearance and embedded in the mucus in the process of destruction.

Altered blood coming from higher portions of the digestive tract is significant, but its demonstration is often difficult. Diagnoses based upon the macroscopic appearance alone are often erroneous, particularly when the feces are not of the typical tarry consistence but are compact.

Confusion with feces containing bismuth is precluded by the great number of crystals of bismuth suboxid revealed in every microscopical preparation. As a rule, only chemical or spectroscopic analysis will demonstrate the presence of hemoglobin.

In the recognition of ulcers or of malignant neoplasms of the stomach and intestine, paramount diagnostic importance has in the last few years been attached to the demonstration of minimal amounts of blood in the feces, the so-called *occult hemorrhages*. To obtain a positive result from blood tests, and this is especially true of the very sensitive Weber-van Deen test, it is necessary to exclude blood originating from food, therefore for several days prior to the investigation meat should not be taken by the patient; and we should also positively ascertain that small hemorrhages from the mouth or the pharynx are not the source of the bleeding.

TEST FOR OCCULT BLOOD

To carry out the Weber-van Deen test which is now popular, a large mass of feces (if the amount of fat is excessive perhaps after preceding treatment with ether) is mixed with water which contains about one-third its volume of glacial acetic acid until the mixture is of fluid consistence, and the hemoglobin is extracted in a test-tube by shaking with ether. To the ether which has been poured off ten drops of a freshly prepared guaiac tincture are added (one part of resin to 25 parts of absolute alcohol) and then (shaking after the addition of each drop) 20 to 30 drops are added of old turpentine oil which has been standing for several weeks in a flat bowl exposed to the air. A blue tint to the ether proves the presence of hematin. In shaking the ether with turpentine we should avoid covering the top of the test-tube with the thumb as the perspiration on the skin may affect the accuracy of the test. On account of the same fallacy in the test the presence of pus also must be excluded.

As to the well known hemin test of Teichmann, I think that, as a rule, too much table salt is used, a very small granule being sufficient; after heating we should wait a long time before examining the preparation so that the crystals may have time to form; finally, for unknown reasons the test is occasionally negative notwithstanding the positive presence of hemoglobin.

Pus cells in large quantities, partly with and partly without a mucus intermediary substance, are rarely found in the feces. Their appearance is, however, of great diagnostic importance and proves the presence of ulcers or a ruptured abscess. If merely isolated round cells permeate the fecal mucus this view is not permissible, and we can at most diagnosticate only an extreme irritation of the intestinal mucous membrane. The impossibility of demonstrating pus cells in the individual case by no means excludes the existence of ulceration, since small clumps in the diarrheic feces may escape recognition or they may have been dissolved by decomposition or the digestive ferments.

In exceptional cases *shreds of tissue* from the intestine, of ulcers, of hemorrhoidal nodules, or necrotic portions of the intestinal wall, especially in the case of invagination and of dysentery, may be desquamated and discovered in the feces. Their origin can be decided only by microscopic examination. They must not be confounded with tough mucus and animal membranes which are the residue of food.

This description of the investigation of the feces is by no means complete. Bacteriologic methods and the detection of animal parasites have not been touched upon. In the chemical aspect of the subject, clinical reasons constrained me to be brief; here much might be added. But it was my primary object to describe fully and especially emphasize the points most important to us as practising physicians, and clearly to portray the method of analysis of the feces which at the bedside will give us the most accurate information.

DIARRHEA, INTESTINAL CATARRH, AND INTESTINAL TUBERCULOSIS

By W. FLEINER, Heidelberg

I. DIARRHEA

The designation *diarrhea* for those disturbances of intestinal digestion in which the discharges become more or less soft or fluid, and also are evacuated more than once during the day, originates from the view that food and drink are propelled very rapidly or almost rush through the intestine. This rapid propulsion through the intestine indicates *increased intestinal activity*; the appearance in the dejecta of constituents of the food points to *insufficient digestion and absorption*; the softer, more fluid composition of the discharges to *insufficient absorption of fluid*, and to *increased secretion* of the intestinal mucous membrane and the glands connected therewith. At all events, in diarrhea intestinal digestion is disturbed; therefore, all diarrhea is a symptom of intestinal dyspepsia.

As in gastric pathology we differentiate dyspepsias from organic gastric disease as purely functional disturbances, so we must discriminate between functional *intestinal dyspepsias* and organic diseases of the intestine. It would, however, be a great error invariably to characterize gastric or intestinal functional disturbances as nervous. Dyspepsia may, here and there, be of purely nervous origin; it is, however, the natural consequence of the condition that we do not sharply differentiate between intestinal dyspepsia and intestinal diseases, for sometimes intestinal dyspepsia is the precursor, sometimes an accompaniment, of an organic intestinal disease. Sometimes it exists independently, or the disease upon which intestinal dyspepsia is dependent has its seat in some other organ than the intestine.

Normally, the processes of digestion in the gastrointestinal canal are adapted to the ingested food. The function of the digestive glands is set in motion by chemoreflexes generated by the quantity and quality of the food and drink, and exactly regulated (Pawlow). The epithelia of the intestines facilitate the propulsion of the products of digestion, and their villi dip into the chyle, just as the roots of plants dip into the earth which furnishes their food, taking from it water, salts and other nutritive products which digestion has made fit for absorption, and which the organism requires for the structure of its tissues, for its growth, its development of force, the rehabilitation of what has been used, and for the formation of heat. The unused remains of chyle, the residue of food, the products of excretion from the liver and the intestine itself, permeated by an enormous number of bacteria, are excreted by the intestine as feces.

The property by which the secreting and absorbing organs accommodate themselves to the nature and amount of the ingested food is, however, not absolute, but shows individual limitation. Therefore, a disproportion between force and amount may readily be brought about, and the juices of digestion and the power of absorption in the intestinal canal prove insufficient for the demands made upon them. The excess of chyle not taken up into the fluids of the body by digestion and absorption then abnormally irritates the small intestine, which attempts to rid itself of its irritating contents by increased peristalsis—diarrhea from the small intestine. In the less sensitive colon there may be compensation, and hypermotility, or diarrhea of the small intestine, may even be checked. True diarrhea, i. e., fecal discharges of soft or thin consistence, only occurs after decided irritation of the colon.

The stimulus for normal movements of the stomach and intestines originates from the contents, but in the intestines also from the chemical irritation of *products of fermentation and decomposition.*

To show the intensity of their effect upon intestinal movement, Boka arranged the *organic (fermentative) acids* of the intestinal canal in th following order: Lactic acid, succinic acid, valerianic acid, butyric aci formic acid, propionic acid, acetic acid, caproic acid and caprylic aci The mildest irritation was produced by lactic acid, the severest by capryl acid. These, as well as caproic acid, even in small doses produced i animals tonic intestinal spasm and decided hyperemia. Moreover, t foregoing acids not only caused diarrhea but, even in small amoun produced catarrh; given in larger doses, decided inflammation of t intestinal tract. In contrast with these the salts of organic acids, even much larger doses, produced no intestinal movements.

Of the *intestinal gases* hydrogen and nitrogen proved to be indiffere while carbonic acid, marsh gas, and sulphureted hydrogen caused sev intestinal movements.

Among the *products of proteid decomposition,* Bokai found that *in* had but slight action and *phenol* none at all, upon intestinal moveme On the other hand, even small doses of *skatol* produced severe perist in the small intestine and rectum, and also gave rise to tonic spas these areas. Skatol at first causes the blood-vessels to contract, but i few minutes this contraction is succeeded by a dilatation of the ves Skatol did not produce catarrh.

These reports indicate the great importance of intestinal bacteria normal and pathologic processes in the intestinal canal. It may, there be interesting to discuss somewhat more minutely the ***relation of bac to the intestine.***

INTESTINAL BACTERIA

At birth the intestinal canal contains no bacteria (Billroth), but each hour of extra-uterine life increases the number of bacteria conveyed into the intestinal canal of the nursling from its surroundings. As a rule, no harm is done the infantile organism by this bacterial invasion so long as the child is nourished by its mother's milk or that of a wet-nurse. The extraordinary sensitiveness of the intestine of the nursling to any other food, to cow's milk and to substitutes for milk, is, however, well known, and the marked tendency of infants to diarrhea, and the great mortality of children in the first years of life from gastric and intestinal disturbances, are usually attributed to the abnormal invasion of bacteria. Very interesting facts were communicated to us by v. Behring in his article "Diarrhea among Calves" (Kälbersterbe), and also concerning the mortality of nurslings in his well known publication "Säuglingsmilch und Säuglingssterblichkeit" (*Therapie der Gegenwart*, Heft I, 1904). In the dysentery of calves which is due to infection with the bacterium coli, and which may also develop in newborn calves by nourishing them with *boiled cow's milk*, according to Joest, the best prophylaxis has proven to be fresh milk from the mother cow immediately after the birth of the calf. Infant mortality can also be reduced from upwards of 30 to 3.55 per cent. by providing the child with a wet-nurse: Therefore, in breast milk, and in the milk of the mother animal, there exists a certain inherent property which protects the nursling and the sucking animal.

In the blood of cattle, v. Behring found isopathically developed antibodies for the most important intestinal bacteria, that is, for the bacterium lactis and bacterium coli. These protective bodies, as also the albumins and globulins of the blood and milk, enter the milk from the blood, and v. Behring, in fact, demonstrated that fresh cow's milk has a very active antibody antagonistic to the bacterium coli, and a weaker one to protect it from the bacterium lactis. The bactericidal property of milk is less than that of the serum, corresponding to the smaller percentage in blood albumin. These antibodies are destroyed by boiling the milk, but not by digestion; they may therefore pass through the intestinal wall of the nursling into its fluids in an unchanged condition. Upon the basis of these experiences v. Behring constructed his new process for the preservation of milk. The addition of formalin, 1 to 5,000 or 1 to 10,000, to the milk, as proposed by him, is said to increase the power of the antibodies.

With the advance of extra-uterine life, the organism itself produces certain protective products, the power of resistance in the intestinal wall increases, and, in consequence, the intestine of the nursling no longer acts like a filter with large pores, to which it has been compared. Verworn ascribes exclusively to the absorbent intestinal epithelia (which act like

Finally, the biliary acids must be considered as antiseptic agents in the intestine. Maly and Emich report that the biliary acids set free from the cholates by the hydrochloric acid of the gastric juice, particularly taurocholic acid, have but little less of antiseptic action than salicylic acid and phenol. Naturally the disinfecting power of free biliary acids is only active while, and so far as, the chyle in the small intestine is of acid reaction. This period, however, is too brief to permit thorough disinfection.

If other bactericidal products should be found in the intestinal juices, we must assume that the varieties of intestinal bacteria which continue to live are those which, in the struggle for existence, feed upon the bacteria which constantly enter the gastrointestinal canal by means of the saliva or with food and drink. The different processes of this struggle are, at present, entirely beyond our knowledge. But the fact of its occurrence is proven by numerous experiments.

Thus R. Schütz, who introduced large quantities of Metschnikoff's vibrios directly into the duodenum of dogs through a fistula, was unable to cultivate this germ from the feces provided the intestinal activity of the animals experimented upon was normal and the intestinal mucous membrane uninjured. Bienstock fed garden earth containing tetanus germs to animals, but was unable to produce tetanus by inoculation experiments. The bacterium coli simultaneously cultivated with diphtheria bacilli in meat bouillon prevented the development of the latter, yet it succumbed to other microbes, for example, to streptococci and staphylococci.

Processes of fermentation and decomposition in the intestine ar strongly influenced by the food, and the physiological and pathologica processes in the intestinal canal and the changes in the fecal discharg are most intimately connected. These conditions may be readily invest gated in small children.

The feces of healthy nurslings resulting from normal breast milk a voided two to five times daily, have an acid reaction, and an acid od which is not unpleasant. They are of a yolk-yellow color, pappy, ar according to the amount, the total solids from 1 to 1.3 per cent. of t dried food. Intestinal decomposition increases when nurslings are nou ished with cow's milk; the feces are of lighter color, of firmer consisten have an alkaline reaction, and the odor of feces or of decomposition. T amount of total solids rises to 2 3.1 per cent. of the dried food. Ferme tation and decomposition in the intestine become more marked when ch dren are nourished [illegible]. In such children the total solids of t feces rise to [illegible] per cent. of the dried food, therefore are five six times as great as those of nurslings nourished with mother's m (Biedert).

Infants who do not enjoy the blessing of mother's milk, and who "artificially" nourished, are greatly injured by over-feeding and overloading the stomach and intestines. If the child's stomach is not relieved by frequent vomiting, and the warning given by profuse evacuations of feces permeated with cheesy clumps and of disagreeable odor is disregarded, severe intestinal disturbances soon appear. These cheesy masses readily irritate the delicate intestinal wall mechanically, producing more violent peristalsis and the premature diarrheal evacuation of constituents of the food not yet absorbed and utilized. Added to this loss of nutrition, however, is the baneful factor that undigested, superfluous masses—the *deleterious residue of food* described by Biedert—promote a dangerous increase of bacteria in the intestine by furnishing a suitable culture media for the generators of fermentation and decomposition already present there, for the saprophytes which enter with the food, and, under some circumstances, for pathogenic bacteria. According to the nature of the food, a surplus of products of fermentation then produces *acid diarrhea,* or an abnormal proteid decomposition, *fetid diarrhea.* Other, often severe, forms of diarrhea which threaten the life of young children by intoxication or infection are produced by definite, more or less specific kinds of bacteria, and are especially influenced by season and climate, particularly by heat.

Adults who have no accurate knowledge or clear conception of the requirements of the body, and who, either from a love of eating or from habit, overtax their digestive organs by over-eating or by flooding them with wine and beer, and who thus fill the lower portions of the intestines with fermentative and decomposing material, suffer, as do young children, from "deleterious residues of food" in the intestine.

For a time the habit of over-distending the normal digestive organs produces no symptoms other than profuse and frequent compact or soft dejecta. This condition, designated by earlier authors as "*copropoesis,*" i. e., increased formation of feces, polycopria, is readily combined with a tendency to diarrhea, which, sooner or later, passes into habitual diarrhea that may continue for years and decades, often without much effect upon the general nutrition of the patient. This significant "*copropoesis*" and the tendency to diarrhea, if disregarded, are frequently followed in the course of time by severe injury to the intestine and other organs; for example, the liver, the kidneys, the heart and the vessels, and this is enhanced by advancing age and by certain occupations, for example, those necessitating a sedentary life. To the diarrhea which assumes a chronic form, flatulency is added, and the inflation of the abdomen, borborygmi in the intestines, and the frequent discharge of gas of a very disagreeable odor, become most annoying. The constantly distended intestine, especially the colon, gradually becomes blunted to irritation always present and loses its tone. The first sign of flaccidity—which precedes long torpor of the intestine—is bloating of the abdomen and the over-distention of individual portions of the intestine or the entire colon from gas (partial or total *intestinal atony*). This flaccidity is not necessarily due to advancing age, for it may occur at any period of life, and an abdomen enormously distended with gas is especially conspicuous among poorly nourished children who suffer from chronic diarrhea.

Heavy food containing little of nutritive products rich in residue, especially of a vegetable nature, also causes overloading and extreme irritation of the intestine. We cannot nourish ourselves with just enough pure food according to its calory value, but require for a sense of satiety, and especially to stimulate peristalsis, the aid of food containing residue. If, however, the fluids of digestion are insufficient to assimilate and absorb a large amount of nourishment which, however, is poor in nutritive products, the undigested residue of the food will cause irritation, and the products of bacterial decomposition will give rise to diarrheic discharges which in the course of time are followed by catarrh and flaccidity.

The habitual overloading of the intestine and the consequent chronic intestinal dyspepsia which runs its course with diarrhea are opposed by an *occasional* form of acute intestinal dyspepsia due to *quantitative* errors in diet. Any excess in eating or drinking may bring this about. More or less severe intestinal disturbances which may also appear without pronounced symptoms on the part of the stomach are relieved in a comparatively short time if the intestinal canal becomes empty by the free discharge of gas and diarrheal evacuations. Without any excess whatever, a *qualitative* error in diet may be made unconsciously, or may pass unnoticed, and the error in nutrition may be recognized only by its consequences. Here individual circumstances, personal susceptibility of the intestine or the peculiarities of its bacterial flora play an important rôle. Some intestines are so irritable that diarrhea ensues on the slightest change in the food, and simple, non-irritating foods, which others always include in the dietary, act like purgatives. I do not refer to fresh fruits, vegetables, cucumbers, sauerkraut and fermenting drinks, which are known to possess laxative properties, but call attention to milk, fo which some persons have an actual idiosyncrasy, and to which adults ar often more susceptible than nurslings.

But the assumption of an abnormal or nervous *irritability of th intestine* will not explain all of the many acute and chronic forms o diarrhea, particularly when the so-called dyspeptic intestinal phenomen appear without an actual error in diet. After the ingestion of foo hygienically suitable, and when the stomach functions normally, insuff ciently digested chyle may be washed into the intestine by fluids, suc as plain water, carbonated water, tea or coffee, taken while eating o soon afterward, and intestinal dyspepsia, abnormal fermentation, the gen eration of gas and diarrhea are frequently the consequences.

Exceedingly often the irritability of the intestine which, even wit ordinary food, causes intestinal dyspepsia and diarrhea, is the consequence of buccal dyspepsia or gastric dyspepsia. A disturbance of digestion in the mouth is due to insufficient mastication and to the incomplete admixture of saliva with the bolus, and occurs in all the affections of the mouth, particularly in caries of the teeth, with deficient dental prosthesis, and, much more frequently, from the bad habit of bolting the food. Even an excellent stomach is incapable of disintegrating large portions of food, nor can the intestine accomplish this, hence it suffers from the mechanical irritation of the fermenting and decomposing foreign body.

The various *gastric dyspepsias* produce intestinal dyspepsia and diarrhea in many ways. *Hypermotility of the stomach,* even when the secretory function is normal, fills the intestine to repletion with insufficiently prepared chyle. When there is a disturbance of gastric juice secretion, or a decrease or absence of hydrochloric acid, as in *achylia* or *apepsia,* the chyle also passes from the stomach into the intestine insufficiently prepared. If, in consequence of atonic conditions of the stomach—either with a normal or pathological secretion of gastric juice—hyperacidity develops from abnormal fermentation, even the upper small intestine simultaneously contains bacteria and large quantities of those acid products of fermentation whose stimulating effect upon peristalsis has been experimentally proven by Bokai. To all this must be added that, with the absence of hydrochloric acid in the gastric juice, the specific generator of pancreatic juice secretion is absent, and this renders intestinal digestion more difficult. In the undigested portions of the chyle excessive fermentation and decomposition take place. After what has been said, the mouth and the stomach may be quite properly designated as protective organs for the intestine. But all intestines do not require this protection, for frequently intestinal digestion is normal, and the state of the nutrition is good; often even constipation is present, notwithstanding these functional disturbances in the mouth and stomach.

Just as in the intestinal dyspepsias which have so far been described, in which products of fermentation and decomposition of the intestinal bacteria produce diarrhea by chemical irritation of the walls of the intestine, so this is also brought about by the products of metabolism of many pathogenic microörganisms.

The greatest irritation to the intestinal wall is caused by the *bacterial toxins* which are recognized as strong poisons. Toxic diarrhea in consequence of poisoning from meat, sausage, fish, mushrooms, potatoes, beans, etc., is more rarely observed than the sporadic forms, those occurring in families or in entire districts, the so-called diseases of the masses. The poisons are of *ectogenous* origin, and find their way into the body with tainted food, for *alkaloids of decomposition* and *cadaveral alkaloids,* so-called ptomains, do not develop under normal conditions of intestinal decomposition in the body. Brieger has found numerous products of this kind in decomposing masses of meat. He observed that some of the bases isolated during the first period of decomposition (monamins and diamins) were physiologically indifferent, and some slightly toxic. After the third day of decomposition, however, more poisonous ptomains or toxins appear, and, on prolonged decomposition, substances of extraordinary toxicity (mydalein, methyl-guanidin and others). Even under pathological conditions, ptomain formation very rarely occurs in the intestinal canal itself, but invariably in *cholera,* and in a peculiar anomaly of metabolism, cystinuria (Neumeister). In pure cultures of the cholera bacillus and of the Finkler-Prior cholerine bacillus, after twenty-four hours, Brieger found pentamethylendiamin, and in older cholera cultures putrescin, cadaverin and methyl-guanidin. Therefore, intestinal autointoxication cannot be so greatly distributed nor so important as Bouchard maintains, although we refer to abnormal processes of decomposition in the gastrointestinal canal many general symptoms of intestinal dyspeptic disturbance, for example, lassitude and depression, headache, nausea and vertigo.

ETIOLOGY

Chemical substances and poisons introduced into the intestine from without irritate the intestinal wall just as do products derived from bacteria, and, by increasing peristalsis and the flow of the intestinal juices (secretion of dilution), produce more or less distressing diarrhea, hyperemia and inflammation.

Most of the milder *laxatives* act only by stimulating peristalsis, espe-
cially peristalsis of the colon; therefore, they free only the colon fro
feces and, at most, give rise to fecal diarrhea. The thin, fluid compos
tion of the dejecta is due to its rapid propulsion through the intestin
and consequently there is diminished absorption of water. If we desi
to induce increased secretion of the intestinal juices, or transudation fro
the blood into the intestine and watery diarrhea, alkaline salts are admi
istered in [illegible] solutions. In spite of the existing osmotic differenc
in pressure, the current of diffusion from [illegible] intestine is only in t
direction of the vascular tract and not in the contrary direction, becau
[illegible] body (O. Coh
heim [illegible] the intesti
[illegible] the fluids of t
body [illegible] and arsenic.
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[illegible] is damag
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[illegible] alkaloids

numerous chemical preparations which cannot here be individually enumerated.

Some of the substances mentioned may also produce diarrhea *by way of the blood*, for example, cathartic acid injected subcutaneously, colocynthin, aloin and others. Mercury, the mercurous chlorid of which is designated mite (hydrarg. chlor. mite), has a more highly toxic action from the blood than calomel from the intestine. Severe intestinal disturbance with superficial epithelial destruction or deeply invading necrosis of the mucous membrane has not rarely been observed from the external use of corrosive sublimate, for example, after vaginal and uterine douches. I have also known chronic diarrhea and colitis to develop in surgeons from the frequent disinfection of their hands with solutions of corrosive sublimate, and to be kept up by this method. The laxative effect of tobacco smoking appears to be due to the fact that nicotin is absorbed into the circulation from the oral cavity or the pharynx, or it may be absorbed by the lungs with the inspired air. Sometimes, however, the effect from tobacco is produced so rapidly—while at other times it does not appear at all in habitual smokers—that we must presuppose a suggestive action.

The diarrheas which occur at the onset or in the course of many infectious diseases are due to the circulation, the intestine showing no pathological change. In this category we include the loose stools observed in septicemia, in pneumonia, in malaria, in influenza and erysipelas, as well as those which take place in cholera and from putrid poisoning. The diarrhea which occurs in nephritis and uremia is the result of urea excreted from the blood into the intestine; in its transformation into ammonium carbonate this is very irritating, and even produces corrosion and necrosis. As a rule, we refer to *nervous influences* those forms of diarrhea in which no dietetic errors or pathologic conditions in the intestine can be recognized. Under normal conditions the digestive processes are controlled only by the gastrointestinal nerves; but these communicate with the pneumogastric nerve as well as with the sympathetic, with the cerebral and peripheral nervous systems; therefore, by these avenues abnormal processes and pathological conditions may derange the intestinal function. These functional disturbances may be motor, secretory, or sensory; they may continue for a long or short time, may recur periodically or become chronic, and may then produce clearly defined clinical pictures, for example, chronic diarrhea and peristaltic unrest of the intestine. In this manner, in the stomach as well as in the intestine, the various functions individually or collectively are abnormally *stimulated* or *inhibited*. This intestinal action may show variations, one function may be increased, another, on the contrary, may be inhibited or even absolutely arrested.

We are most familiar with the effect of sudden and great psychical stimulation upon the motor function of the gastrointestinal canal, and such disturbances in function are attributed to psychical irritation of the nerve tracts which increases peristalsis or to psychical paralysis of the nerves which inhibit intestinal motion. Not infrequently, by *fright*, *anxiety*, or *fear*, the contents of the stomach are thus propelled into the intestine more rapidly than usual, and the intestines are more rapidly evacuated.

As to the effect of these involuntary and irresistible intestinal discharges, the laity in Germany have improvised characteristic designations for nervous people and cowards. Innumerable examples of this might be quoted from daily life; I shall limit myself to calling attention only to the increased peristalsis, the desire to evacuate the bowels, and even the diarrheic discharges (which actually take place even with repetitions) from which many persons of both sexes suffer during the excitement of a journey, while in company, before an appointment, preliminary to making a speech, in anticipation of going to court, etc. An auto-suggestive effect is produced by the mere sensation of traveling in a railroad car without toilet facilities, of being in a house or in company, etc., where it may be inconvenient to attend to the bowels.

Diarrhea due to the irritation of the skin by *cold*, and produced *reflexly*, is nervous diarrhea only in a certain sense, for example, when it follows chilling of the feet, a wetting, sitting upon a cold seat, or taking a cold bath; it may also arise from the diseased condition of organs adjacent to the intestine, especially the internal genitalia of women, and in genito-urinary diseases of men. During menstruation diarrhea dependent upon affections of the female genitalia shows periodic aggravation. There are also diarrheas which depend upon organic diseases of the spinal cord, such as the intestinal crises with diarrhea which periodically appear or periodically show aggravation.

In actual, chronic, uninterrupted, continuous diarrhea, varying onl in intensity, I advise great deliberation before making a diagnosis o nervous diarrhea. I do not deny the occurrence of chronic diarrhea havin a nervous or psychopathic foundation, but I believe it to be very rar In many cases of this kind, if on strict diet, clinical observation and th regular examination of the feces will reveal the starting point of th irritative condition somewhere in the intestine, or external to it. Th sequels of a former perityphlitis, a pericholecystitis, or other local inflam mation of the peritoneum, must here be borne in mind, as well as als tuberculous ulcers in the rectum, polypi situated higher, frequently i women old and neglected lacerations of the perineum or prolapse, an in children enterozoa.

Whether the very persistent and exhausting diarrheas in Graves' di ease, like the tachycardia, depend upon nervous hypermotility of the gastr intestinal canal or whether they are due to autointoxication cannot wit our present knowledge be positively decided.

SYMPTOMS

As a rule, the symptoms in diarrhea are quite uniform; those which have preceded or may follow the diarrheal discharges may show many variations, and those observed in nervous persons may bear a very peculiar stamp.

In some cases the patients complain only that the intestinal discharges are more frequent than usual, that their consistence is abnormally soft, or more or less fluid. Not rarely the preceding intestinal dyspepsia produces symptoms. A short time after the usual meal, often even during the meal, a distention of the abdomen is noted and is exceedingly unpleasant. This is followed by noticeable motion in the intestine, often only in the form of peristaltic unrest, such as gurgling and rumbling, but also often as increased peristalsis accompanied by slight pain. Other painful sensations may now and then be felt in the abdomen. These are caused by irritating food substances or abnormal digestive products, and indicate either local irritation of the mucous membrane—cutting pains, griping pains—or spasmodic contractions of the muscular layer of the intestine occurring in portions of the intestine already irritated, or they are due to the distention of isolated intestinal coils by gas or feces. Both spastic contraction of the intestine and great distention of the same—the former being a muscular pain, the latter a peritoneal pain—produce "belly-ache" or "griping," and when this becomes intense it is designated "colic." The paroxysmal, periodic appearance of these pains is characteristic; slight at first they increase in severity and become most intense; the cessation or disappearance of the pain, which resembles "labor pain" and is often compared with it, is also typical. In the intervals between the attacks the pain may cease and the patient seem restored to complete health; sometimes, however, in these intervals between the marked colicky attacks, a greater or less degree of sensitiveness remains until the irritating intestinal contents have been completely evacuated.

According to their cause, these colics have been designated *wind colic* (*colica flatulenta*) or *fecal colic* (*colica stercoralis*). Usually, however, these forms are combined. In these colics, the pain ameliorates as soon as gases or feces are discharged, the attacks are less frequent and the recurrences less severe. Inversely, however, they increase in intensity when the passage of gas or feces is prevented. The gases discharged may, according to their nature, produce a burning sensation which may continue for some time. This is also true of the feces, particularly the thin ones mixed with undigested fluids which corrode the anal mucous membrane.

These colics may be of such intensity as to cause even adults to writhe; in debilitated persons they sometimes cause syncope, and in children they frequently lead to general convulsions, even to loss of consciousness.

If we remember the physiology of intestinal movement, and particularly the fact that local contraction results from a local irritation of the intestine above the point of irritation, and that in consequence of this an annular constriction forms while the intestine below the point of irritation becomes flaccid and dilated, the frequency of invagination in small children, whose intestine, as is well known, is much more irritable than that of adults, is easily explained. As a matter of fact, during intestinal colic in children invagination of the small intestine and of the colon frequently occurs and is chiefly due to marked local irritation; often the colicky pains are the consequence of such invaginations. Spontaneous recovery no doubt takes place provided such invagination is but slight; in other cases intussusception of greater or less extent supervenes, and by constriction or occlusion of the intestine may threaten life.

Aside from complications on the part of the stomach (the intolerance of which toward any food or drink may cause debility, as in cholera nostras) the effect of diarrhea upon the general strength and nutrition depends chiefly upon the number and size of the evacuations during the day. The discharge of deleterious masses from the gastro-intestinal canal may be beneficial, but since not these alone are discharged but also a quantity of nutritive substances, and especially much of the fluid of the body, all profuse diarrheas mean a loss of fluid to the organism. In children and debilitated adults, therefore, diarrheas produce a rapid loss of strength because such persons possess but little reserve material. After profuse diarrhea the turgescence of the skin rapidly disappears; the skin becomes flaccid and dry, the face hollow, the nose pointed, and the eyes are sunken in their cavities. Corresponding with the loss in water through the intestine, the amount of urine decreases, and the blood-pressure in some cases is so much reduced that the pulse becomes imperceptible. In severe cases in adults who evacuated from two to three liters of thin fluid feces in twenty-four hours, I have seen complete anuria which only disappeared when repeated subcutaneous infusions of normal salt solution were given or when the extreme thirst led the patients to supply the fluid loss by drinking. As a rule, a *restitutio ad integrum* rapidly occurs provided the diarrhea is not too long continued and is not due to severe anatomical lesions.

Nevertheless, even in acute diarrhea, the loss of fluid with the numerous, watery discharges is more or less injurious. The feeling of exhaustion, the cardiac asthenia in diarrhea, has been referred to the lowered blood-pressure from loss of fluid, also cramps in the calves and the appearance of tonic spasms in unilateral and symmetrical muscles of the extremities, of the trunk, and of the face, also in the muscles of the pharynx, larynx and eyes; in fact, the entire picture of tetany, including all its other symptoms, has been referred to inspissation of the muscular and nervous tissue from losses of water (Kussmaul, Fleiner). Other authors attribute these severe musculo-nervous disturbances to gastrointestinal processes of intoxication. Their etiology, however, has not yet been decided, as no similar toxic products which have this effect have yet been proven to exist in the gastrointestinal canal of patients with diarrhea.

In most cases of severe diarrhea there is a loss of weight even if the condition persists but a few days. This, however, is rapidly compensated if no severe organic disturbances are present, because the loss of weight is chiefly dependent upon the loss of water. In prolonged or chronic diarrhea, the condition is different. Here, in the course of time, added to the loss in water is the absence of resorption, therefore an insufficient ingestion of food, and, in consequence of this, a utilization of the reserve fund of fat, glycogen and albumin of the body, therefore, under-nutrition and emaciation. In children and adults the latter may be of alarming extent; moreover, during this period of under-nutrition, in children the general growth of the body and also of individual organs ceases, particularly that of the teeth and the bones, and a predisposition to other maladies, especially to infectious diseases, rickets, scrofulosis and tuberculosis, is established. Besides extreme emaciation, adults with severe forms of chronic diarrhea often show a peculiar form of glossitis, apparently dependent upon atrophy of the epithelium at the tip and borders of the anterior half of the tongue, and atrophy of the papilla upon the dorsum of the tongue. The surface of the tongue is free from coating, it is smooth and glistening like the mucous membrane of the mouth, and very sensitive to chemical and mechanical irritation. Small children, and adults greatly weakened by disease, particularly the senile, not rarely succumb to profuse diarrhea. In consequence of the great loss of fluid and the excessive limitation of absorption, chronic diarrhea sometimes leads to severe forms of anemia, to cardiac weakness the result of fatty degeneration of the heart, to hydremia, and occasionally to albuminuria which finally causes death.

Middle-aged men who eat heartily and hastily, and who are accustomed to drink a great deal, withstand chronic diarrhea remarkably well for years and even decades. In spite of three, or many more, soft, diarrheic evacuations daily, their general nutrition does not suffer; on the contrary, they increase rather than lose in weight, and they are even indifferent to a decided degree of flatulence, until, from atony of the intestine, constipation, flatulent and fecal colic, colitis or severe complications on the part of other organs (for example, the heart or the kidneys) and atheromatosis, etc., are superadded.

The reaction of intestinal disturbances upon the *nervous system* varies in its manifestations; as from the stomach so from the intestine every abnormal irritation may be conducted to the central organs of the nervous system by centripetal tracts in the pneumogastric and sympathetic nerves. In persons of neuropathic tendency, or those who are debilitated and anemic, there are intestinal neuroses of the pneumogastric nerve which, similar to the gastric form, disturb cardiac activity and the respiration, and produce sensations of constriction, a feeling of anxiety, and vertigo. This so-called intestinal vertigo, which, as a rule, appears suddenly during a diarrhea of brief duration, and is sometimes synchronous with the fecal movements, may come in severe attacks or in a milder, but more protracted form; the latter is more likely to be induced by constipation than by diarrhea. There are, however, cases of paradoxical constipation notwithstanding profuse, even diarrheal discharges, and these conditions are frequently associated with such attacks of vertigo. In connection with intestinal dyspeptic disturbances, the pulse invariably becomes irregular and intermittent. To this peristaltic unrest of the intestine, which may be purely nervous or psychical but is usually due to abnormal intestinal irritation, general nervous excitement due to intestinal action is added, as well as noises of a gurgling character which at night prevent sleep, or cause the patient to awaken in a state of nervous anxiety which sometimes takes the form of definite fears (worms, frogs, etc., in the abdomen). Closely allied to these terrors is the hypochondriac depression, originating in sensations produced by peripheral irritation of the sympathetic, and referred by the consciousness to fear, which constantly centers attention on those organs and parts of the body which are threatened. Such individuals are actually tormented by their sensations and imaginations; they, however, make all those about them miserable, not less so their physician, or, more correctly, their physicians, who are annoyed much more than is necessary by their patients with intestinal hypochondriasis.

Migraine and hemicrania intestinalis, or hemicrania dyspeptica, ar often produced by diarrhea; sometimes, however, migraine ceases with th appearance of diarrhea. The conditions are similar with hyperesthesia an neuralgia of other localizations, as well as vasomotor disturbances whic in a certain sense, are related to intestinal dyspepsia, such as urticari acute and chronic erythema, and acne-like eruptions of the face, wheth this relation be established by the nerve tracts or by the circulation, reflex action and irradiation or by intoxication.

DIAGNOSIS

The diagnosis of an intestinal affection must not be based upon the r ognition of one particular symptom of the disease—for example, diarrh constipation, colic, or hemorrhage—but a number of problems must be ta into consideration. In diarrhea, for instance, the cause and the seat the disturbance must be determined, and we must decide whether a me functional disturbance—hypermotility, hypersecretion, or diminished sorption—is the basis of the malady, whether this functional disturban the expression of pathological changes in the intestinal wall, and whe these are of substantive character; therefore, whether they are of intestinal origin, are dependent upon disturbances or diseases of other organs, or are due to general constitutional conditions. If the latter, the organs directly connected with the intestine must, above all, be investigated; first the oral cavity and the stomach, then the liver and the pancreas, later the peritoneum and the organs related to it, and especially the female genitalia, then the nervous system, the composition of the blood, the organs of circulation, and, finally, the kidneys. The clinical examination of the entire body and its individual organs is absolutely necessary to determine the origin and the action of processes of disease in the intestines. Of these we actually get but imperfect knowledge, and this chiefly by an *examination of the feces*, which occupies the same position in the diagnosis of intestinal diseases as the examination of the gastric contents in the diagnosis of gastric diseases, the examination of the sputa in the diagnosis of respiratory diseases, or the examination of the urine in renal diseases and in disturbances of metabolism.

Examination of the gastric contents and the intestinal discharges has not as yet attained such popularity in practice as examinations of the urine and the sputum. The methods are more complicated and difficult, nevertheless the pith of a voluminous literature of investigations of the gastric contents and their value in practice is infinitesimally small and simple. Contrasted with a pure, scientific *coprology*, those methods of examination of the feces which permit a positive clinical diagnosis and furnish clear therapeutic indications are just as simple. First among these methods is *coproscopy*, simple inspection of the feces. When the unaided eye is not sufficient the lens and the microscope should be employed, or even chemical reagents. Quantitative analysis of the feces is only rarely necessary in practice, although its value in solving scientific and physiologic questions in the laboratory cannot be overestimated.

Like everything else, coproscopy, the inspection of the feces, requires practice, and, if we expect it to yield results, inspection must be not only occasional, but regular and continuous until the diagnosis becomes absolutely clear or the patient ceases to be under observation. In order to secure any reliable data from the examination of the feces, we must have a knowledge of the patient's previous diet. When the findings are doubtful, the question will arise whether other foods, drink, or drugs have been taken.

For example, let us consider a profuse, diarrheic stool in which large and small particles of solid food of various kinds may be recognized with the naked eye: This form of diarrhea has long been known as *lientery*. The investigation is made in order to decide *which constituents of the food have been carried or propelled rapidly through the intestine, and what abnormal products the intestine has contributed to the fecal admixture.* The size and amount of the undigested particles of food evacuated lead us to suppose that an abnormally large amount of food was eaten, that it was hastily swallowed, i.e., was insufficiently masticated. Consequently, we examine the teeth of the patient, order a reduction in the amount of the food, and insist that in the preparation of the food in the kitchen it be more finely divided by hashing or other mechanical means, or that this be done during eating either by artificial or natural mastication (a masticator). Coproscopy, repeated the next day; may reveal a normal condition. Therefore, there was no actual intestinal disturbance in the previous lientery but the cause was merely a poor condition of the teeth, incomplete mastication, or overeating.

If in a given case, *notwithstanding a prescribed diet,* undigested particles of food appear in the feces, this variety of lientery admits of quite a different interpretation. We now investigate the character of these unabsorbed constituents of the food. If the fecal discharges are thin this is sometimes very easy, for the solid constituents may be extracted by the aid of a pipette or a small stick. It is sometimes advisable to receive the latter in a wire net or fecal sieve (Boas). Necessarily, compact feces must be carefully broken up in a vessel with water by a pestle.

The most valuable elements in our food are *albumin* (meat), *fat,* and *carbohydrates,* while the residue of vegetables is of subordinate importance Hence, when we find with the naked eye or with the microscope fragment of food in the feces we know that we are dealing either with an albumi (or *meat*) *diarrhea* or a *fat diarrhea,* that is, *albumin,* or *meat,* c *connective tissue lientery* (*proteorrhea*) or a *fat lientery* (*steatorrhea* Accordingly, with the relatively rare appearance of starch in the feces, w designate the affection as amylum lientery or amylorrhea (v. Oefele).

The results of the physiology of digestion teach us that albumino products are digested in the stomach by pepsin and hydrochloric acid, a in the intestine by the proteolytic ferment of the pancreatic juice (trypsi while fat, which is only slightly absorbed in the stomach (Volhard), chiefly absorbed in the small intestine by the steapsin of the pancrea juice. The carbonated alkali of the pancreatic juice, the bile, and fluids of the small intestine combine with free fatty acids, and, by giv off carbonic acid, fatty alkalies or soaps develop at any stage between molecules of neutral fat which have not as yet been split up. Thu fine emulsion of microscopically small fat globules is formed in the in tine. The digestion of carbohydrates begins in the mouth by the ac of the ptyalin of the saliva during mastication. If the stomach cont have an acid reaction, amylolysis ceases in the stomach; but it be again in the small intestine by the action of the ptyalin of the panc juice.

The most important carrier of albumin is meat. From what has previously stated we know that the presence of undigested meat i feces indicates a disturbance of gastric or pancreatic digestion in the intestine, and a careful investigation of the functions of the stomach in the usual manner is advisable. The result will show whether the disturbance is gastric or pancreatic, and whether we should prescribe hydrochloric acid and pepsin or a preparation of pancreas. To complete the technic of fecal examination, it must be mentioned that the passage of undigested meat is evidence of impaired gastric and pancreatic digestion. Connective tissue, according to Kühne, is digested in the stomach; *the finding of shreds of connective tissue in the feces therefore indicates a lack of gastric digestion*, HCl-anacidity, achylia gastrica, or apepsia. Muscle fibers are digested in the small intestine by trypsin; hence the macroscopic recognition of remains of muscle or well-retained transverse muscle striæ by the microscope indicates a *disturbance of digestion in the small intestine*—deficient trypsin digestion. The passage of undigested remains of milk in the form of cheesy flocculi is subject to the same interpretation.

With a little practice, an enormous increase of fat in the stool may be certainly recognized by the oily, creamy consistence, the pale, grayish, yellow or grayish white fatty gloss, by the fact that the feces float in water, and their behavior when pulverized (a membrane showing a play of colors). Under the microscope such feces show unsplit, neutral fats in globules, and fatty acids and soap needles in profusion. On analyzing these findings, we are forced to consider *disturbances in the flow of bile and in bile production*; therefore, diseases of the liver and biliary passages (occlusion of the common bile duct, jaundice), derangement of the pancreatic secretion, and actual diseases of the small intestine. Doubt can only be removed by clinical observation combined with therapeutic measures (pancreatic preparations) and chemical examinations of the urine and feces, which cannot be considered in this article.

The carbohydrates of the food, provided this is properly prepared in the kitchen and thoroughly masticated, are usually well digested. The discovery of food particles containing starch indicates, therefore, an extremely rapid propulsion through the intestine. Since, however, the carbohydrates which escape digestion and absorption ferment and rapidly decompose in the intestine, we are justified in concluding from the presence of large quantities of acid products of fermentation (acid reaction of the feces) or a great volume of gas in the bowel or in the feces (perhaps A. Schmidt's fermentation test) that there is deficient carbohydrate digestion in the intestine. The absence under the microscope of the iodin reaction is not always conclusive; digestion of starch may be deficient and the reaction for starch in the feces be very slight provided the undigested starch has been destroyed by fermentation.

We must search for the cause of poor digestion of starch in the improper preparation of the food, in imperfect mastication and insufficient admixture with the saliva, also within the bowel itself (intestinal fermentative dyspepsia).

The treatment of acute and chronic diarrhea will be considered in the chapters relating to intestinal catarrh.

II. INTESTINAL CATARRH

Diarrhea and intestinal catarrh running its course with diarrhea present in common the disturbances in function previously described. Often, therefore, they are confounded with one another. This error, however, is not so great as it may appear; for between diarrhea and intestinal catarrh there is often a difference only of degree, and simple diarrhea may precede true intestinal catarrh, may continue as a functional irritation, and cause a catarrhal inflammation of the intestinal mucous membrane. The chief difference between diarrhea and intestinal catarrh consists in a pathologic change in the intestinal wall which may be clinically recognized from the admixture of mucus to the diarrheic stools.

ACUTE INTESTINAL CATARRH

This is one of the most common maladies, and occurs in man at every age, but especially in small children, in the aged, and in the debilitated who have little power of resistance. Acute intestinal catarrh is usually a primary and substantive affection, more rarely it occurs secondarily and symptomatically in the course of previously existing diseases.

Etiology.—The most frequent cause of intestinal catarrh is the abnormal nature of the intestinal contents which immoderately irritate the intestinal mucous membrane, either mechanically or, more frequently, chemically. Those causal factors which have been enumerated for the development of diarrhea may also be of etiologic importance in intestinal catarrh Among the normal products of fermentation and decomposition of th intestinal contents which are of great importance in peristalsis, we men tion also those which, according to Bokai's investigations, not only pr duce diarrhea, but also spastic contraction of the muscles of the intesti and catarrh and inflammation of the gastric mucous membrane. The sa dietetic indiscretions, the immoderate eating of food and drinking of fluid or the unsuitable nature of these and their injurious effect upon the bo —briefly, all errors in diet of either a quantitative or qualitative nature may in the one case produce only diarrhea, and in another give r to intense, catarrhal, intestinal symptoms. Local and periodic con tions which increase the virulence of bacteria may favor the ectogen decomposition of food, so that the toxic action of the abnormal produ of decomposition not only results in functional irritative symptoms also in pathological changes in the intestinal wall. In the intestine it a foreign vegetation introduced with spoiled food may replace the mal intestinal flora or even exceed this in growth, and by abnor decomposition also generate products in the intestinal contents—therefore endogenous—which damage the intestinal mucous membrane. This *chyme infection* of Escherich is the opposite of *intestinal infection*—the direct damage of the intestinal wall by bacteria and other specific pathogenic agents.

Many infectious diseases begin with the appearance of gastrointestinal catarrh, which, as in the case of enteric fever, cholera, and dysentery, may be attributed to the accumulation and development of specific pathogenic microörganisms and their effect on metabolism. In all probability, the majority of the pathogenic microörganisms are capable of producing infectious intestinal catarrh; this is certain, at least, of *staphylococci* and *streptococci*. Similar in action to bacteria are *amebæ*, *coccidia* and *infusoria*, which are common in the tropics and find entrance to the intestine by means of stagnant or decomposed water. Upon entering the mucous membrane they cause the death of the epithelium by a poison that they develop, and even in the deeper layers of the intestinal wall they produce inflammation and necrosis which, in the severer forms of enteritis, lead to ulceration. Just as abscesses develop in the liver by means of the portal vein circulation in dysentery and in some forms of ameba enteritis, so may metastatic inflammation and septicemia follow infectious intestinal catarrh. The conditions are the reverse in other infectious diseases which are not localized in the intestine; for example, in puerperal fever and other septic processes, also in the *dyscrasias* which are the consequence of some infectious diseases (tuberculosis, syphilis, malaria and pyemia, as well as in carcinomatosis and in uremia) by producing an abnormal composition of the blood or, on the contrary, the excretion of definite toxic-acting products into the intestinal canal, which there produce intestinal catarrh.

In the forms of disease last mentioned, the mucous membrane of the entire digestive tract may be the seat of catarrhal inflammation, for in catarrh due to abnormal intestinal contents the localization and the distribution of the catarrh depend upon the extent of the *materia peccans* into the intestine, the length of time it retains its noxious properties, or where it assumes this virulent nature.

An acute gastric catarrh distributes itself within the upper small intestine, not, as was formerly assumed, by the extension of the inflammatory process from the gastric mucous membrane to the duodenal mucous membrane, but by the propagation of the same irritating, deleterious agents. The lower portions of the intestine may be exempt when chyle, which is still abnormal in the duodenum and in the jejunum, becomes innocuous by further dilution with the digestive juices, or by dilution with a large amount of fluid which has transuded into the intestine, or when it is coated with mucus. On the other hand, abnormal processes of decomposition may frequently be generated in the deeper portions of the small intestine or in the beginning of the colon, in regions where the conditions are more favorable for the existence of microbes than in the upper portion of the small intestine or in the stomach.

Except when caused by abnormal fermentation and decomposition, catarrh of the colon is most frequently caused by the abnormal dryness and hardness of the feces, by their prolonged retention, or by their accumulation in large masses. Owing to peculiar local and functional conditions, definite areas of the colon are predisposed to catarrhal and inflammatory affections; above all, the cecum, the flexures at either side of the abdomen, and the sigmoid flexure. The same is true of the rectum; but, besides accumulated feces in the ampulla, various kinds of ingesta produce catarrh of the mucous membrane, as do also *enemata* which irritate thermically, *nutritive enemata* which irritate by their concentration or by the decomposition of food products which they contain, and ***drugs*** which are injected into the rectum. Occasionally gonorrheal virus produces a severe catarrh of the rectum. Refrigeration must frequently be considered as a cause of acute intestinal catarrh, and also, finally, intestinal parasites and foreign bodies which find their way into the intestine.

Secondarily or symptomatically intestinal catarrh arises in other pathological, structural changes of the intestinal wall, in many ulcers, in inflammation of the peritoneum, and in local and general circulatory disturbances which affect the intestinal canal. Abnormal displacements and the fixation of individual intestinal coils, also invagination, torsion, and incarceration, lead to stretching and compression of the mesenteric vessels, and consequently, to local circulatory disturbances, changes in the mucous membrane, and intestinal necrosis. In all of these conditions it is true tha intestinal catarrh, as such, is of minor importance.

Pathology.—In acute intestinal catarrh the pathological lesions ar often out of proportion to the severity of the clinical symptoms, especiall in the cases which run a rapid course and in which severe intestinal infe tions or intoxications have caused a speedy death. In these instance chemical and bacteriological investigations lead to more accurate concl sions than the autopsy findings.

When a catarrhal enteritis is present the intestinal tract only exce tionally shows inflammatory change; as a rule, merely isolated portio are diseased, some more, others less, while extensive areas of the intesti canal are often entirely exempt. The diseased mucous membrane may covered with a slimy, or, more often, a serous, or, at other times, a mu purulent and turbid exudate from desquamated epithelia and pus, and t may sometimes be of even a hemorrhagic nature. The mucous membr itself is flaky or diffusely reddened and swollen, is more markedly ir trated than normally, often showing hemorrhagic infiltration or pern tion by small hemorrhages, particularly in the crests of the mucous m brane folds.

Many intestinal catarrhs are characterized by swelling and redness of the follicular apparatus (enteritis follicularis). The solitary follicles then form reddish or grayish white prominent nodules, and on the agminate glands (Peyer's patches) are grayish white or gray elevations surrounded by a hyperemic area, and permeated by groove-like depressions.

Microscopically, the mucous membrane is markedly hyperemic, and frequently shows a very defective epithelial coating as a post mortem phenomenon. The epithelia still present have undergone a mucoid and destructive change, the supporting tissue and the submucosa, according to the intensity of the inflammation, give evidence of serous transudation and small cell infiltration, while, on the other hand, the muscularis is slightly or not at all altered.

In accordance with the localization of the inflammation, we differentiate pathologically between duodenitis, jejunitis and ileitis, a typhlitis (appendicularis and typhlitis cecalis), colitis (even sigmoiditis) and proctitis. Clinically, these differentiations are impossible.

As a rule, in the milder forms of acute intestinal catarrh complete recovery occurs. After the removal from the intestinal canal of all deleterious products the inflammatory irritation ceases as well as the pathological secretions, the products of inflammation are absorbed, and the epithelium which has been destroyed is replaced by regenerative proliferation of the epithelial cells retained in Lieberkühn's crypts.

If the inflammatory irritation is long continued, or if it recurs rapidly and often, chronic intestinal catarrh may develop from an acute form. More intense irritation leads to severe inflammation of the mucous membrane and of the deep layers of the intestinal wall, to necrosis and to ulceration. Severe infectious and toxic forms of enteritis not rarely result in death, and they form the chief cause of the enormously great mortality of infants.

Symptoms.—Among the clinical symptoms of acute intestinal catarrh the most common are intestinal motor, sensory, and secretory irritative phenomena: *Diarrhea, pain* and the admixture of *mucus* to the feces. In the severe infectious and toxic forms of intestinal catarrh to these must be added the *general symptoms of infection and of intoxication* which not infrequently may be of very serious nature and extremely dangerous to debilitated persons.

Among the symptoms, diarrhea and the pain of various kinds so often combined with it have been described. I have also emphasized that an intense irritation of the colon is a typical characteristic of the diarrhea. In fact, there are many forms of catarrh of the small intestine which cannot be recognized except by those who make it a rule in all gastrointestinal affections to subject the feces to regular and minute examination. Duodenal catarrh, for example, may be masked by the symptoms of gastritis, with which it is usually associated until the appearance of jaundice, which originates from the duodenum. Catarrh may also be of the jejunum and of the ileum; in fact, catarrh limited to the small intestine is frequently unrecognizable because it has so few diagnostic points of support. It is true that it may run its course with marked disturbance of the constitutional condition and local difficulties, but diarrhea is absent, or present for only a few days, when the colon also becomes irritated and the seat of catarrhal inflammation. Nothnagel has described as *jejunal diarrhea* a condition in which feces are evacuated which are of the same composition as the contents of the jejunum: Odorless masses with unaltered constituents of the food and unaltered bile, and permeated with mucus. Such diarrheas occasionally occur. Much more frequently, however, catarrh of the jejunum and catarrh of the upper ileum run their course without diarrhea, at least for a time. Not rarely we observe in such cases irritative symptoms which usually precede diarrhea—increased peristalsis, rolling movements, borborygmi and pain—but the diarrhea does not occur for some time. Notwithstanding this—probably in consequence of the too rapid propulsion through the small intestine of the undigested remains of food—abnormal fermentation generates in the large intestine an increased gas formation which causes some distress, and which may be clinically recognized until, after more or less time, diarrhea supervenes. In the evacuated feces insufficiently digested fragments of food will probably be found, but often, at the onset of diarrhea, no unaltered bile. Only in catarrh of the small intestine and colon combined, which runs its cours with frequent diarrhea, is bile again passed in an unaltered condition. I such feces we sometimes find, besides unaltered bile, also other digestiv juices, or, at least, their ferments, pepsin and trypsin. On exposure to th air their bilious tint occasionally changes and becomes greenish, or leel colored.

Abdominal pain is common in most cases of intestinal catarrh. This usually dull, sometimes of a burning or cutting character, and may continuous or increase and decrease periodically. This may spread ov the entire abdomen, or may be localized in the region of the navel, esp cially if the small intestine, in particular, is implicated. Acute catarrh the colon appears to cause much greater pain than that of the small int tine; the pain is localized in the ileo-cecal region, in the flexures of hypochondriac regions, and in the sigmoid flexure, or is often confined the transverse colon or may extend throughout the entire course of colon.

Pains of the greatest intensity may be caused by a simple *muc membrane catarrh of the appendix*; very frequently this suggests foration; however, peritoneal phenomena and fever do not follow, anc in such cases, as a precaution, the appendix is resected, the slight ch in the mucous membrane of the excised appendix is surprising. It not be out of place to emphasize the fact that the vermiform append predisposed to extremely painful colics which appear upon the slightest irritation.

Diarrheal discharges from the large intestine also cause periodic attacks of colic which are frequently of great intensity. As already stated, these are attributable to spastic contracture of the annular musculature of the intestine or to the great distention of certain portions of the intestine by gas or feces (incarcerated gas, wind colic), while the non-spasmodic intestinal pains are to be ascribed to chemical irritation of the sensory nerves of the intestinal wall. In many intestinal catarrhs pain is absent; the diarrheic stools are forcibly ejected from the bowel.

Proctitis.—Acute proctitis is accompanied by peculiar pain which in mild cases shows itself by a more or less distressing sensation of burning, often by ineffectual attempts to evacuate the bowels, and, in severe cases, appears in the form of spasmodic tenesmus. Not infrequently—although but little fecal matter is discharged—the mucous membrane of the rectum to a greater or less extent protrudes from the anus as a tumor, or tumor-like mass, almost ectropic. Occasionally, as in colica spastica, spasm of the anal sphincter appears and may continue for some time, giving rise to severe pain, and preventing evacuation. Often in acute proctitis mucus alone is discharged, sometimes of a glassy or shreddy character, occasionally also in peculiar globular or cluster-like masses; when there is severe tenesmus, besides mucus blood also invariably appears in the dejecta. Often hemorrhagic mucus, or blood alone, is noted without ulcerations being necessarily present in the rectal mucous membrane.

In acute intestinal catarrh the general systemic disturbances vary extraordinarily. Robust persons who have no fever very often pass through an attack without being confined to bed; the evacuations, which often amount to ten or twenty during the day, are very distressing, as well as the feeling of soreness about the anus and the lassitude which is attributable to the great loss of fluid and the impossibility of taking sufficient nourishment, but, in a few days, the difficulty passes away. In severe cases there is a sensation of extreme illness which renders the patient incapable of attending to his work, and confines him to bed; so fulminant do the symptoms appear that a severe infectious disease or an intoxication is supposed to be present until the diagnosis becomes clear.

Fever is more commonly observed and is also higher in children than in adults; the onset and course, however, conform to no distinct type. In the toxic forms of acute intestinal catarrh, the temperature is apt to be subnormal.

Other symptoms frequently observed as accompaniments of dyspeptic disturbances, such as headache, lassitude, nausea, anorexia, coated tongue, thirst, an eruption of herpes about the lips, urticaria, pains in the limbs, also cramps in definite muscle groups, and cramp in the calves, are noted also in acute intestinal catarrh. In severe cases typical attacks of tetany and delirium are now and then observed. In children the nervous disturbances of acute enteritis are particularly severe and, at the onset of the disease, tubercular meningitis may be simulated.

Corresponding in ratio with the losses of water by the copious bowel discharges, at the height of an attack of acute intestinal catarrh the excretion of urine is very deficient, and the daily amount voided is much reduced; often anuria may last for a day or even longer; during this time blood-pressure is decidedly lessened, the pulse is small and often irregular. With the reestablishment of diuresis, albumin may often be found in the urine as well as casts, both, however, only temporarily.

Upon examination the patients, particularly children, are found apathetic and apparently seriously ill. As a rule, very little can be demonstrated objectively except the frequent diarrheic discharges at the onset of the disease. Enlargement of the spleen has now and then been observed in infectious catarrh. The abdomen is usually unchanged, at times somewhat retracted, at other times distended. Through the thin abdominal walls we occasionally perceive the motions of the intestine, or they may be felt with a palpating hand, or rumbling is heard in the abdomen and portends another intestinal evacuation. As a rule, the entire abdomen is sensitive to pressure, but it may be so in only limited areas; gurgling is readily produced in the ileo-cecal region. Profuse accumulations of fluid and intestinal contents here and there cause dulness upon percussion. The comparatively rapid change in the symptoms will, however, soon show how erroneous is the assumption of a peritoneal exudate or an ascites.

In no other malady is the destructive action of the affection so distinctly apparent, and in relatively so brief a time, as in severe acute (gastro-)enteritis. In severe cases striking changes may take place between one visit and another. A blooming child may be changed to a pitiable object, the previously full, round face may be emaciated, a formerly strong heart may be flaccid and weak. Just as rapidly, however, a change for the better may supervene when the profuse discharges cease and copious draughts of fluid have quenched the thirst of the body and its tissues.

Course of the Disease.—In adults the course of acute intestinal catarrh is usually favorable, provided there is no severe alimentary intoxication. Unfortunately, at the onset of an acute enteritis we do not always know that it is the primary effect of an infectious disease—occasionally the latter is, in fact, the case, under which circumstances only in the further course of the disease do the characteristic symptoms of specific infection, influenza, articular rheumatism, enteric fever, or cholera, appear. If, however, there is no reason to suspect these affections, the prognosis in adults is always favorable. In a few days the affection, whose severity is usually limited to the excessive number of bowel evacuations daily, has run its course, and although convalescence may be somewhat protracted the duration of the disease is rarely more than one or two weeks. Only in little children is acute enteritis, particularly gastroenteritis, a severe disease which greatly influences the general mortality.

In persons of any age severe acute intestinal catarrh not rarely leaves an irritative condition of the colon which expresses itself by a tendency to diarrhea, and may persist for a considerable time.

Diagnosis.—The diagnosis of acute intestinal catarrh is, as a rule, very easy; but the localization of the catarrh, and, in some cases which run their course with severe constitutional symptoms, the decision of the question, whether the disease is an infection or intoxication, may be difficult.

In practice, usually, no line of demarcation is drawn between diarrhea and intestinal catarrh, and, indeed, the designation intestinal catarrh appears to be the more popular one for the cases under consideration. The chief characteristic of catarrh, however, is the admixture of mucus with the feces, and this cannot be overlooked if the investigation of the feces is at all thorough.

We regard mucus as a pathological secretion, the presence of which in a given case clearly indicates that the limits of a purely functional disturbance have been exceeded, and that pathological changes have taken place in the intestinal wall; namely, catarrh and inflammation. *The differential diagnosis between diarrhea and intestinal catarrh can, therefore, be made only by coproscopy.*

Mucus is a glycoproteid which upon splitting becomes a proteid and a carbohydrate. This splitting may be produced chemically (by boiling with a 5 per cent. hydrochloric acid solution) after which the albumin body, as well as the carbohydrate, may be demonstrated by reagents. It takes place in the stomach as well as in the small intestine; therefore, *mucus is digestible.*

Consequently sputum that has been swallowed, mucus from the pharynx, from the esophagus, or from the stomach never reappears in the feces; therefore, mucus found in the feces must originate in the intestine. But mucus is also digested in the upper portion of the small intestine; in the colon, however, it is certainly not. Hence it is possible that mucus originating from catarrh of the upper small intestine disappears wholly or entirely in digestion, provided it is not too rapidly propelled into the lower portion of the intestine and voided with profuse diarrheal discharges.

An admixture of mucus with the feces, like the more or less intimate admixture of blood, indicates a somewhat high point of origin. Flocculi of mucus intimately combined with feces, and bile-stained, denote catarrh of the small intestine. This sign is, however, not absolutely certain, for in severe diarrhea unaltered bile may pass through the entire intestinal canal and be readily recognizable in the feces. In doubtful cases, Gmelin's test or Schmidt's corrosive sublimate test is decisive for unaltered biliary coloring matter. This reaction shows an abnormally rapid propulsion through the intestine of all those constituents of the feces which are stained with unaltered biliary coloring matter (and which become green with Schmidt's test).

While a microscope is often necessary to detect small mucus flocculi (and partly digested ones?) from the upper small intestine, mucus from the lower small intestine, also that which comes from the colon, even when intimately admixed with feces, can always be recognized by the naked eye. If only the descending colon, the sigmoid flexure and the rectum are diseased, the mucus masses originating from these areas may easily be detached from the feces; they will rarely show staining with bile. In acute catarrh of the rectum and the descending colon pure mucus is sometimes discharged. An admixture of blood with the mucus or feces is very frequently found in acute proctitis running its course with severe tenesmus even when there is no ulcerative process in the rectum.

The quantity and composition of the mucus admixed with the feces varies greatly; between the finest flocculi, delicate membranes, and large shreddy membranes, all intermediate stages may be observed. Amorphous mucus gives to many diarrheic stools a gelatinous composition; sometimes amorphous mucus is discharged alone as a glassy mass. The uniform admixture of small threads of mucus with diarrheic stools gives to them a choppy appearance (thread-worms).

In the descending colon and rectum the mucus sometimes appears in the form of bands or tubes corresponding in shape with these portions of the intestine. A peculiar globular or grape-like appearance of the mucus, which to the inexperienced sometimes becomes a diagnostic enigma, is acquired in the haustra of the colon or the ampulla of the rectum (in chronic catarrh more frequently than in acute, especially after convalescence has set in) when the feces which are propelled by peristalsis toward the anus take from the mucous membrane its mucus coating and roll it up. Admixture of mucus with the feces does not take place in the lower portions of the intestine; the feces then are covered with mucus—actually enveloped in it.

Microscopic examination of the mucus will protect us from a possible confusion with the remains of food, and at the same time it reveals the intensity of the catarrhal inflammation; this can be estimated by the numbers of white (and, rarely, of red) blood-corpuscles admixed with the mucus. A plentiful admixture of white blood-corpuscles to the mucus gives this a purulent character which may frequently be recognized macroscopically.

The mucus often shows a hemorrhagic discoloration, even with only superficial inflammation of the intestinal mucous membrane. Greater admixture of blood to the intestinal discharges almost always indicates ulcerative processes in the intestine or rupture of continuity of the mucous membrane in the rectum (fissures, abrasions, hemorrhoids). Here it need only be mentioned that profuse hemorrhages, even without ulcerative processes, may take place in the gastrointestinal canal; for example, in hepatic cirrhosis and in thrombosis and embolism of the mesenteric artery.

The causes of endemic and tropical diarrhea, of many acute and chronic catarrhs, and of inflammations of the intestinal mucous membrane are to be found in the protozoa in fresh intestinal mucus or that which has been kept warm. Some forms of *dysentery* belong to this category, also *ameba enteritis, coccidia* and *infusoria diarrhea.* In his article, "Dysentery and Ameba Enteritis," which I should here like to refer to,[1] G. Hoppe-Seyler has described these conditions. But I cannot here enter upon the bacteriologic finding of the specific agents of many infectious diarrheas and intestinal inflammations, neither can I enumerate the parasites of the intestine, the existence of which can be determined by an examination of the feces.

Treatment.—The treatment of diarrhea and of acute intestinal catarrh must be instituted with a search for the cause. If it has been found that abnormality of the intestinal contents has produced the affection *the first indication in the treatment is the removal of the deleterious intestinal contents.*

As a matter of fact the action of the organism is based on the same principles: Diarrhea, produced reflexly, is an attempt on the part of the over-irritated organ to relieve itself, and this is further promoted by the increased excretion of fluid and the inappetence, also reflexly produced, which prevents the placing of additional labor upon the digestive organs, but gives them rest; thus a reserve power of resistance is produced. In fact, many diarrheas and catarrhs are self-limited and naturally run a rapid and favorable course; for this reason we should not interfere with the organism in its attempt at self-aid: *Primo die quiescere satis est; neque impetum ventris prohibere* (Celsus).

We aid this endeavor of the body by rest in bed and the administration of non-irritating drinks which immediately cleanse the stomach and the upper part of the small intestine and, after absorption, also the tissues of the body, especially the blood and the kidneys, thus compensating for the loss of fluids and the diarrhea by maintaining diuresis. By washing out the colon from below a large quantity of its contents can be more rapidly evacuated than by the diarrhea, and thereby we limit the absorption of abnormal products of decomposition, and give the irritated organ longer periods of rest, even if we do not always by these means succeed in rapidly controlling the diarrhea.

Warm infusions of herbs which contain ethereal oils (phenol, anise, chamomile, or peppermint) long enjoyed a great reputation in diarrhea, for the reason that they taste better than warm water, and as carminatives apparently have a mild action; but they may well be substituted for by warm mineral water with a small addition of table salt, by weak warm or cold tea, and, in some cases, by dilute rice water, barley water, oatmeal water or albumin water. When these drinks are to be taken for several days the last named deserve preference, as they simultaneously contain a minimum of nourishment.

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[1] See "Modern Clinical Medicine," "Infectious Diseases," D. Appleton & Co., 1905, p. 681.

The quantity of fluid that is to be administered depends upon individual conditions; it should be sufficient to quench the thirst, but should not be given in excess.

For intestinal irrigations, besides weak infusions of peppermint, chamomile, anise, etc., the most suitable fluids are saline solutions one-half to three-quarters per cent., under some circumstances with the addition of 0.5 to 1.0 of salicylic acid to the liter of fluid. Other disinfectants may be used for this purpose; some of them are not very effective as the solution must be very weak in order not to injure the intestinal epithelium, and in choosing these remedies their absorption in the rectum and colon must be considered. The efficacy of the washing depends largely upon the temperature of the fluid, which should be of the same temperature as the body, and the *quantity* employed must depend upon individual circumstances. I advise that the patient be placed in the left lateral posture, under some circumstances the pelvis to be raised, and as much of the lukewarm fluid to be introduced with low pressure (one-half to one meter) as can be borne without discomfort. As soon as there is a desire to evacuate the bowel we must suspend the irrigation and the patient should be permitted to discharge the fluid injected. In children from 200 to 500 c.c., in adults from 1 to 1½ liters and more may thus be used.

According to the indications of the case, in diarrhea we may irrigate the intestines morning and evening, or even three times a day; for example, in acute diarrhea combined with great tenesmus. In other instances a single washing of the intestine in the morning or in the evening will suffice. In so-called *nocturnal diarrhea,* which invariably occurs at night and, as a rule, in the second half of the night or early in the morning, it is advisable to give a chamomile injection in the evening, shortly before supper, or half an hour after going to bed. Such evening injections are, at the same time, the most effective remedies for peristaltic unrest of the intestine which prevents or interferes with sleep.

The best instrument to employ is an irrigator or glass funnel with a conveniently long rubber tube; but syringes may also be employed i a piece of rubber tubing 20 to 30 cm. in length is inserted between th syringe and the rectal bulb to allow for a possible recoil or jerk o the syringe, and to prevent trauma to the rectum. Care is necessar in the choice of an anal tube; the best are those made of hard rubber o glass which have an olive-shaped bulb and are well and smoothly rounded off, and the rubber rectal tubes to be attached should have fenestra that have been smoothly rounded off. *The high introduction of longer tubes into the intestine is only advisable when the sphincter will not contract, and when we desire to dislodge an obstruction with the rectal tube;* for example, in a retroflexion of the uterus or in the case of perimetritic exudates. Even in the so-called *high* intestinal injection, a high introduction of the rectal tube is unnecessary; the flow of the fluid into the intestine is promoted much more by the position of the patient than by the introduction of a long tube: The knee-elbow position, the dorsal decubitus with the shoulders low and the pelvis high, perhaps even the placing of the patient upon a modern operating table which permits the maximum high position of the pelvis.

Occasionally we thoroughly cleanse the rectum and the sigmoid flexure, sometimes even the left flexure of the colon or the entire colon, by irrigating the intestine with a tube and funnel, similarly as in gastric lavage, raising and lowering the funnel and occasionally, after emptying the funnel, adding more fluid. The intestine, however, is more susceptible than the stomach, and the observance of, at least, the same precautions as in washing the other organ is necessary.

If the injurious intestinal contents cannot be removed by washing from below, it is advisable to try mild laxatives. The number of remedies to choose from is great, therefore individual indications may be considered. Young children bear calomel in small doses (0.01–0.03 several times daily) very well. In adults several (2 or 3) large doses (0.1–0.2) given at short intervals are sometimes of great value in diarrhea, although even with these doses[1] we must reckon with the frequent intolerance of mercury. In doubtful cases a good dose (20 to 30 grams) of castor oil is given, or rhubarb, cascara sagrada, laxative tea or lemonade (citrate of magnesia), a teaspoonful of Glauber salt, Epsom salt, sodium subsulphate or Carlsbad salt dissolved in about a quarter of a liter of water.

The *second indication* in the treatment of acute diarrhea and intestinal catarrh is to produce non-irritating intestinal contents. As a rule, we should be in no haste to bring about such a change. Fluids that are easily absorbed, water, tea, thin meat broth, rice water, and various mineral waters with a small addition of brandy or red wine are sufficient until hunger reappears or weakness becomes noticeable. In the choice of foods the results of the examination of the feces must be considered, and at first those foods must be avoided which coproscopy shows to be

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[1] I particularly desire to emphasize that the belief that calomel disinfects the intestine is erroneous. Schütz proved in his experimental animals that by calomel —in contrast with castor oil—the bactericidal properties of the intestine were inhibited, and Strasburger found more bacteria in diarrheic stools after calomel than in those produced by castor oil.

CHRONIC INTESTINAL CATARRH

Chronic intestinal catarrh, like the acute form, may appear as a primary and substantive affection. Frequently, however, it is either secondary or of a symptomatic nature.

Etiology.—The causes of idiopathic chronic intestinal catarrh are, as a rule, identical with those which produce acute catarrh except for the difference that the baneful agents mentioned here act not only once and intensely, but that this effect is produced repeatedly or continuously. It happens by no means rarely that the chronic form develops from acute enteritis; that, therefore, also a single and transitory but active poison, an indiscretion in diet, severe refrigeration, or an infection, may be the cause of chronic intestinal catarrh. Above all, I desire to refer to the chronic catarrh of the colon which persists after tropical diarrhea and tropical enteritis. To mention an example from the present time, our soldiers who returned from China without having had severe forms of dysentery (although quite a large number had persistent irritation of the colon which in the cases observed by me proved to be chronic colitis) showed impairment of health for a long time although they did not become chronic invalids. More frequently chronic intestinal catarrh sets in, without an acute stage of onset, as the consequence of oft-repeated and continuous injury to the intestine, by improper food and an injudicious mode of life, by the prolonged use of powerful purgatives, and by constitutional anomalies.

"*Morborum fere omnium causa est stomachi infirmitas*" is particularly true of the different forms of chronic intestinal catarrh. Not only does weakness of the stomach impair the bowel, but also that factor which has damaged the stomach. In this connection the poor condition of the teeth, their insufficient use in mastication, the incomplete admixture of the food with saliva, and the bolting of food must be again referred to. We must also consider all the functional disturbances and organic diseases of the stomach which influence the chemical preparation of the gastric contents, above all, gastric dyspepsia, achylia, and apepsia which run their course with deficiency in hydrochloric acid and pepsin To these must be added chronic catarrh of the stomach, hypermotilit which causes the chyle to be too rapidly propelled from the stomach int the intestine, and gastric atony, in which condition the chyle of th stomach undergoes an abnormally acid fermentation, and thus enters th bowel. Even gastric ulcer, which, by HCl-hyperacidity and hyperpepsi makes gastric digestion so rapid, and also by HCl-hyperacidity reflex stimulates pancreatic digestion, may induce chronic colitis as a compl cation if the chyle (which should be wholly absorbed and thus leave b little nutritive material in the intestine for the bacteria of fermentati and decomposition) accumulates in the colon, and if constipation, whi almost invariably accompanies gastric ulcer, then irritates the mucous membrane. Necessarily, however, none of the previously mentioned gastric disturbances need secondarily damage the intestine, for, to a certain extent, and frequently for a long time, intestinal digestion may compensate for derangement of gastric digestion.

The same is true of those disturbances originating in the liver and the pancreas which I have already mentioned.

Pathology.—Pathologico-anatomical changes in the structure of the intestinal wall, and to a less extent tuberculous, dysenteric, typhoid and carcinomatous ulcers, the cicatrices (arising from the former) which produce stenotic tumors, and peritoneal adhesions which limit peristalsis, abnormally fix the individual intestinal coils, or produce changes in their position, and thus hinder the propulsion of feces; these changes are almost invariably associated with intestinal catarrh, which, while sometimes limited to the vicinity of the primary lesion, occasionally may be distributed over extensive areas of the bowel. In fact, any condition may be combined with an impediment to the propulsion of feces, whether this be due to organic changes in the intestinal wall, to functional disturbance of intestinal peristalsis (habitual constipation), or whether it is caused mechanically by pressure or external torsion upon the bowel (uterine and ovarian tumors, hypertrophy of the prostate, peri- and parametritic, perityphlitic, pericholecystitic, perigastric or pericolitic adhesions, indurations and pseudo-membranes), and may give rise to chronic intestinal catarrh. We must mention also as causes of chronic intestinal catarrh the *circulatory disturbances* which produce chronic venous hyperemia of the intestinal mucous membrane. Among the more common of these conditions is chronic intestinal catarrh from abdominal plethora which is the consequence of a sedentary mode of life. Furthermore, intestinal catarrh is common as the consequence of stasis of the portal vein circulation, in hepatic cirrhosis and constriction of the portal vein, in valvular disease of the heart and chronic myocarditis, in chronic diseases of the lungs and pleuræ, and in chronic simple and tuberculous peritonitis.

In conclusion, parasites now and then produce chronic irritation and catarrh of the intestine.

The *pathological changes* produced by chronic intestinal catarrh are chiefly manifested in the mucous membrane, but the submucous tissue and the muscularis are often also implicated, much more frequently revealing inflammatory hyperplasia than a degenerative atrophy.

The mucous membrane is hyperemic and shows cellular infiltration; consequently it is swollen, and covered with mucus or muco-purulent masses. In some areas it is grayish red, livid, rusty brown, or it may show a slaty gray pigmentation. The submucosa is thickened and infiltrated; serous transudation is evident. The connective tissue is proliferated; the gland ducts are often elongated, and occasionally denote cystic degeneration from constriction. The muscularis is sometimes hypertrophied and thickened. In differentiating these conditions, however, the contraction of the intestine must be borne in mind, for, if this is increased, it readily simulates hypertrophy, and an abnormal distention of the bowel may be easily mistaken for atrophy of the various layers of the intestinal wall. At the ileo-cecal valve a chronic inflammatory thickening of all these layers, especially of the musculature, may bring about stenosis of the lumen, which resembles pyloric stenosis due to chronic inflammation and may be the cause of invagination.

The question of intestinal atrophy, especially atrophy of the intestinal mucous membrane, has been much discussed of late since Gerlach, Heubner and others designated intestinal atrophy as a frequent post mortem phenomenon. Whether a vital pathologic atrophy of the mucous membrane can be absolutely denied must be decided by renewed investigations as soon as possible after death.

Fatty degeneration of the muscular fibers, more conspicuous in the longitudinal than in the circular fibers, has been noted now and then in chronic intestinal catarrh, but, as a rule, it is rare; it has been described by Kussmaul, Maier, and E. Wagner. There are but few isolated observations of sclerotic and fatty degeneration of the intestinal nerves, the ganglia and fibers of Meissner, and of Auerbach's plexuses.

The functional disturbances of the intestine produced by chronic intestinal catarrh are prominent in the clinical picture; associated with these are disturbances of nutrition and blood formation as well as other constitutional symptoms.

Symptoms.—While in acute intestinal catarrh diarrhea is characteristic as a motor irritative factor, in chronic intestinal catarrh this is rare, and constipation is the rule. In explanation of this it has been assumed that, under the influence of pathologic processes in the intestinal wall in chronic catarrh, the muscularis of the intestine suffers functionally by the production of *atony,* or from an organic damage by degenerative *atrophy.* But with chronic catarrhal inflammation frequently no change can be observed in the musculature of the intestine. Therefore neither atony nor atrophy of the intestinal muscularis will explain the constipation in chronic catarrh. Nothnagel has assumed that the cause is sluggishness of the intestines, a lessening of the *automatic* activity of the nervous apparatus of the intestinal wall, due to the pathologic processes of chronic catarrh, especially to chronic venous hyperemia. But this decrease in automatic activity does not simultaneously decrease the irritability of the nervous apparatus of the intestine to external influences. This is evident from the periodical and spontaneous occurrence of brief or more prolonged diarrhea, produced by errors in diet, drugs or fecal accumulation. Besides, in chronic intestinal catarrh due to local irritation, not infrequently large or small portions of the intestine may assume a state of spastic contracture in which tonic spasm of the muscularis is indicated by severe pain (colica spastica). This spasmodic contracture may cause the retention of fecal masses in the greater portion of the colon (spastic constipation).

When constipation is absent in chronic catarrh of the intestine, abnormalities of the bowel movements may be observed, and this is true even when the bowels are apparently regular. These abnormalities are shown by the composition of the feces, which are of pappy or semi-fluid consistence with an admixture of mucus, by the evacuation of undigested particles of food, and by an abnormally fetid odor in consequence of decomposition of the pathologic secretions of the bowel.

In other cases of chronic intestinal catarrh constipation and diarrhea alternate in a certain regularity and periodicity. Without apparent cause, after one or several days of constipation, diarrhea occurs spontaneously, continues from one to several days, then ceases, and constipation reappears. Nothnagel has compared this alternation of constipation with diarrhea to the variations of the Cheyne-Stokes respiration, and assumes that, in the cases in question, constipation was originally present and persisted until by stasis of the feces in the colon a condition of irritation was produced which resulted in diarrhea. The irritation ceased after the complete evacuation of the intestine, and constipation reappeared.

With these exceptions, *intercurrent* diarrheas are usually due to some deleterious agent which, by irritating the intestinal wall, increases the intensity of the catarrh; often nervous influences are the cause. *Prolonged* diarrhea, on the other hand, indicates a complication of the intestinal catarrh with ulcerative processes.

Now and then I have observed in chronic intestinal catarrh a peculiar abnormality in the fecal evacuations, which resembles the condition in ischuria paradoxa. A movement of the bowels occurred daily, even regularly, the feces being soft, and the patients never complained of constipation.

Nevertheless, fecal masses were retained in the intestine and gradually increased so that they formed large fecal tumors. Years ago, when I was an assistant in the Pathological Institute, I held an autopsy upon a case of this kind and found in the colon a fecal mass weighing 4 kilograms. In the sigmoid flexure, where the largest fecal tumor was situated, there was an extensive pressure necrosis of the mucous membrane, also pressure ulcers one of which had perforated. Up to the time of death the patient in question had daily evacuated thin stools which had been regarded as sufficient. Among other cases of this kind, my most recent one is interesting. It occurred in a medical student who had severe colicky pains and a most offensive diarrhea, the thin fecal discharges being sometimes involuntary and soiling his bed and body linen most disagreeably; he was treated in the Clinic, and had also been treated elsewhere unsuccessfully, with opium. When I examined this unfortunate man, I found a fecal tumor of about the size of a child's head bulging from the rectum, the sphincter of which was open. Manually I removed as much of this as was possible without anesthesia. Five days later this procedure—although diarrhea was still present—had to be repeated because on the removal of the fecal tumor another formed. After this the pains and the accompanying disturbances of the bladder ceased, and the bowels were regulated by injections of oil. Field has described such a condition under the name of cumulative constipation, and Courtade as latent constipation. From its analogy to ischuria paradoxa, I believe that this might well be designated as *constipatio paradoxa,* that is, a retention of feces notwithstanding existing diarrhea. A very intelligent patient who suffered from this paradoxical retention of feces quite aptly designated her disease as "*constipating diarrhea.*" The condition reminds us of some cases of chronic intestinal catarrh and constipation in which liquid bowel discharges follow the ingestion of fruit, the use of some laxatives and mineral waters, and also occur in a mineral spring treatment, but do not relieve the patient by a satisfactory emptying of the bowel.

The *pain* in chronic intestinal catarrh is usually insignificant; nevertheless there are *irritable forms,* particularly of catarrh of the colon—colitis muco-membranacea—in which the patients suffer greatly from pain and persistent colic, similarly as in nervous mucous colic. In catarrh of the colon the pains radiate to the small of the back, to the hips and to the thighs, and very often to the entire back, and are lessened or entirely relieved by the evacuation of the accumulated fecal masses. The latter is true not only of some cases of sciatica but also of other neuralgias.

In other instances the patients are greatly annoyed by periodic or long-continued *disagreeable sensations* in the abdomen, and these sometimes engender a peculiar, melancholic state of depression. These sensations are chiefly localized in the hypochondria or the region of the flexures of the colon, but are also noted in other areas of the abdomen and the pelvis (the designation hypochondriasis, now employed only for certain depressing psychical conditions, was originally applied to some localized affections in which the colon was presumed to be especially implicated). These disagreeable sensations appear to be due to movements in the colon which are manifested in the way described; that is, they are increased after the ingestion of food, before and after a passage from the bowels, while frequently the patient is at ease so long as the intestine is at rest, i. e., while there is constipation. Besides the formation of othe chemically irritant products of fermentation and decomposition, abnorma gas production is probably the cause of increased intestinal unrest, and rumbling, gurgling, or splashing sounds are heard not only by the patien but by those about him. The patients feel as though the fluid content of the intestine were forced from one part of the abdomen to anothe or as if the intestines were filled with gas which could find no vent. Patients with disease of the intestine who have become hypochondriacs often torment themselves with strange fancies as to the cause of these sensations and sounds; these imaginations correspond with the remedies to which they resort to ameliorate the condition, especially the dietetic penance.

In undernourished persons with a thin, flaccid abdominal wall, peristaltic movements of the intestine are often visible and palpable. In these cases inflation of the abdomen from gas is rarely absent; often it is general, at other times limited to individual portions of the intestine. The passage of flatus usually brings relief, but the opposite condition is most unpleasant to the patient. The accumulation of gases in the bowel causes much discomfort, disturbance of sleep, a feeling of unrest and anxiety, a sense of constriction, arrhythmia and intermittent pulse, cardiac palpation which is often combined with precordial distress and difficulty in respiration. Not infrequently intestinal gas becomes incarcerated in dilated coils of intestine, and produces intense colicky pains (wind colic from overdistention of the intestinal wall).

In other cases of chronic intestinal catarrh the abdomen is retracted, the colon in certain areas or in its greatest extent is contracted, may then be felt as a band, and is painful to the touch. Upon palpation the sigmoid flexure may usually be felt as a cylindrical band which rolls under the palpating finger. Sometimes only one portion of the colon is contracted while other parts are flaccid or even meteorically distended.

The *nutrition* always suffers in chronic colitis, but especially when the small intestines are markedly implicated and less so when the disease is chiefly confined to the colon. Catarrh widely distributed throughout the small and large intestines causes extreme emaciation and debility. The best known forms of this are atrophia infantum, pedatrophia, or tabes mesenterica, the sequel of chronic intestinal catarrh in children in which, in contrast with the enormously distended belly, the patients are emaciated and withered to a skeleton, and with their sad, deeply sunken eyes, small, senile faces, and dry, discolored, withered skin they present a pitiable picture of suffering and human helplessness.

In adults this general disturbance of nutrition is rarely so marked as in children; but I have repeatedly known middle-aged persons to succumb to intestinal catarrh and chronic diarrhea which were the results of tropical enteritis. The dirty gray discoloration of the skin in some persons with chronic constipation which accompanies intestinal catarrh is typical. This "*constipation tint*" may be readily differentiated from that of anemia, cachexia, jaundice, and also from Addison's disease. To what alterations in the composition of the blood it is due cannot always be determined with the ordinary methods of blood examination. Dyscrasic intoxication may be presumed, but cannot always be certainly proven.

Anemic conditions are found in chronic intestinal catarrh, especially when the property of absorption in the small intestine is diminished. While, however, in this condition of hyponutrition we are dealing chiefly with *oligemia,* in colitis there is a destruction of red blood-corpuscles, probably due to toxic products of decomposition, particularly when the condition is accompanied by constipation. Inspissation of the blood with an apparent increase of the erythrocytes to 6,000,000 or more and a corresponding increase of hemoglobin I have observed at various times in prolonged diarrhea due to chronic gastrointestinal catarrh; tetany as a complication was noted in only one case. Severe progressive anemia which was formerly referred to atrophy of the intestinal mucous membrane is comparatively rare in chronic intestinal catarrh. In such cases Eisenlohr described degeneration of a dyscrasic nature in the posterior and lateral columns of the spinal cord which was very similar to that I found in Addison's disease.

THE URINE.—In chronic intestinal catarrh the condition of the urine depends mainly upon the relation of intestinal absorption to intestinal excretion. In severe diarrhea the daily amount of urine voided may fall to that of transitory anuria; constipation does not decidedly influence the amount. Albuminuria occurs in disturbances of intestinal digestion, and is not infrequent in abnormally increased decomposition in the colon; in fact this latter condition plays a rôle which is not to be underestimated in considering the etiology of nephritis as well as that of many diseases of the liver and the blood.

Disturbance of gastric digestion is rarely absent in chronic intestinal catarrh, often being the cause of the latter, and frequently its consequence. The gastric dyspepsia resulting from intestinal catarrh appears to cause greater subjective disturbance than a primary gastric affection. In many cases it is difficult to say whether the digestive symptoms, such as loss of appetite, a disagreeable taste, a coated tongue, pressure and fulness in the abdomen, discomfort after eating, eructations, flatulence and nausea, are to be attributed to the stomach or to the intestine. I believe a coated tongue is more frequently due to intestinal than to gastric disturbance, and most often results from constipation.

Various *nervous symptoms* are to be regarded partly as reflex phenomena from the intestine, and partly as due to the absorption of toxic substances formed by abnormal processes of decomposition in the intestine. To this category belong the oft recurring headache, the migraine and other forms of neuralgia, vertigo, psychical depression, disinclination for mental or physical exertion, a feeling of weakness and lassitude, drowsiness or insomnia, arrhythmia and intermittent pulse, tachycardia, as well as tetany and the other spasmodic conditions which are the consequence of constipation and which are especially observed in small children and feeble easily irritated persons.

The appearance of fever in chronic intestinal catarrh indicates a complication with ulcerative processes and the implication of the peritoneum in the inflammation.

Diagnosis.—The diagnosis of chronic intestinal catarrh can be based only upon the results of the examination of the feces, for the history, the subjective symptoms, and the general phenomena, even the clinical investigation of the abdominal organs, are not conclusive; *the demonstration of mucus in the feces is alone decisive.*

The fundamental laws for the examination of the feces and their application have been described in the preceding sections of this article. They are generally applicable, and therefore may be employed in chronic catarrh of the intestine, at least in that form running its course with diarrhea. To complete my description, however, *I must enter somewhat more minutely into the condition of the feces in chronic intestinal catarrh which runs its course with constipation.*

In this affection the bowel movements are often found to be immoderately large. From this fact we may differentiate copropoesis or polycopria, provided these large masses are regularly voided for a long time; where this is not the case, but small portions and then occasionally very large ones are passed, the evacuations are insufficient and there is consequently fecal accumulation.

When the fecal masses voided are invariably large—large in comparison with the ingested food—there is poor assimilation, insufficient absorption in the small intestine, and profuse excretion of digestive juices and pathologic products from the intestinal wall. Accurate investigation of the feces for undigested remains of food permits the recognition of their nature and the seat of the disturbance, and simultaneously indicates, as has already been stated, the advisability of testing the function of the stomach, and also, perhaps, the glycogenetic function of the liver (alimentary glycosuria). A decision as to the *proper quantity* of the feces, aside from their form and composition, requires much practice (repeated weighing of the total solids of the feces, which is alone decisive, is impracticable) and presupposes an exact knowledge of the previously ingested food, its quantity, composition and preparation. We have here, it is true, a constantly varying but, nevertheless, in its composition and nutritive value, an average diet (already referred to in my "Text-Book of the Diseases of the Digestive Organs"), a similar form of nutrition for all patients under observation until, from the requirements of the individual case, deviations from, additions to, restrictions in, or substitutes for this normal diet become necessary. As the feces of all patients are examined separately from day to day, and are preserved for subsequent and necessary tests, it is easy to determine pathologic change in a bowel movement, any alteration in the form, the consistence, or quantity, also any abnormal admixture; even individual peculiarities may be at once recognized. I believe it to be important to resort to coproscopy in every case, not only once or occasionally, but regularly and until in a given case we have arrived at a definite conclusion in regard to the digestive processes, not only the intestinal but also the gastric, not only the digestibility of the chief articles of food but also of a great number of foods. This method of clinical observation, of the study of the ingestion of food, and of the examination of the excreta, which, in the main, I learned from my revered master, Kussmaul, and which I have elaborated systematically, has been for years of such service that the use of Schmidt's test diet to test the function of the intestine has become unnecessary. My method has the additional advantage that it can be adhered to for a long time, i. e., as long as necessary, while the other method is practicable for only a few days. I do not, however, mean to disparage the importance and value of Schmidt's teachings, and advise the employment of his test diet whenever the study of the nutrition and the investigation of the digestive processes cannot be carried out in the manner I have indicated. I also advise the physician not to be content with a single examination, or a single analysis, of the feces in order to make a diagnosis. The demonstration of connective tissue, meat, fat, or mucus in the feces aids us materially in differentiation, just as does the demonstration of albumin or sugar in the urine, or of bacilli in the sputum. But, as a matter of fact, such demonstration points out only where and how subsequent clinical researches are to be begun and further developed.

By a fecal examination we are enabled to recognize, in the given case, whether the chronic catarrh is localized in the small intestine or in the colon. Most frequently the colon alone is attacked, or the colon simultaneously with the lowest portion of the small intestine.

From the appearance of the intestinal discharges, as well as from the subjective symptoms, various types of chronic (colon) intestinal catarrh may be differentiated: Flaccid, atonic or asthenic forms, and nervous, irritable forms. The first usually run their course with constipation; in this atonic constipation the bowel movement is dry and compact, and consists of adherent clumps or cylinders of immoderately large caliber, also of isolated, irregularly formed particles of varying size, and of fecal balls from the size of a nut to that of an apple. These fecal clumps are no always formed in the rectum (in the ampulla of which they may attain their greatest bulk) or the flexures, but often much higher, in the colon or even in the cecum. In chronic catarrh of the colon, running its course with atonic constipation, the abdomen is, as a rule, uniformly distended. O the other hand, when the entire colon is atonic and filled with gases an feces it is asymmetric, and only partially so when but a portion of th colon is diseased. If the abdominal walls are thin, the accumulated fec masses may be felt in the flexures or the cecum, and also in other po tions of the large intestine, as doughy tumors, in rare cases as har globular or cylindrical tumors, and in the colon they may frequently displaced toward the anus. These atonic forms of constipation and catarrh of the colon are found at all ages, not rarely even in children. Occasionally the impacted fecal masses may cause very painful attacks of colic.

The irritable forms of chronic catarrh of the colon are characterized by a tendency to diarrhea and colic. But many cases of irritable catarrh of the large intestine run their course with constipation of that type I have described as *spastic constipation*. In these cases, an irritation affecting the motor function of the muscularis of the colon, a spastic contracture which implicates large portions of this organ, causes the retention of the compact fecal masses, and constricts and compresses them; or the subsequent peristalsis in high portions forces the compact mass through the spastically contracted portions of the intestine. In this spastic constipation —which may also occur without colitis—the composition of the feces is extremely characteristic. The masses are cylindrical and of small caliber, often only of the thickness of a lead-pencil or of the little finger; sometimes they are very long, and in the chamber they assume a spiral shape like the shell of a snail. Sometimes, however, they are so short that their total amount appears to be wholly insufficient. Balls of small caliber, masses resembling the feces of sheep, are sometimes formed in the haustra but are not always characteristic of spastic constipation, for they may be discharged simultaneously with fecal cylinders of large caliber, or may be superficially embedded in these.

In some cases of colitis the types of constipation which have been described appear side by side, and atony and spasm in different areas of the colon may alternate.

MUCOUS COLIC.—The severest form of catarrh of the colon, and at the same time the most irritable, I shall designate as *colitis muco-membranacea*. The views of physicians in regard to this common and stubborn disease have for a long time been far asunder, as is best proven by Whitehead's report: Of 120 authors who have written concerning it, less than 6 have given it the same name. Some lay the greatest stress upon the intestinal symptoms, others upon the nervous phenomena. Cruveilhier, Trousseau, Habershon, Leube and others emphasized its association with other inflammatory affections of the colon, especially with chronic catarrh; v. Leyden, Guttmann and others assume that the condition is due to a specific inflammatory process similar to that which is generated in croupous chronic bronchitis (Litten) and in dysmenorrhea membranacea (Wernich). In contrast with this, many other authors believe the affection to be a neurosis of the bowel, probably a secretory neurosis accompanying other general neuroses. Nothnagel, who was later supported by Ewald and many other adherents, especially emphasizes that the affection chiefly attacks nervous and hysterical women and hypochondriacal men, these presenting the clinical picture of colitis muco-membranacea. In accordance with this conception, Nothnagel proposed that we discard the names colitis and enteritis for this affection, and substitute the designation *colica mucosa, mucous colic.*[1] In attempting to relieve or cure colitis membranacea, while correcting the general nutritive disturbances and regulating the intestinal functions, I have so frequently seen the accompanying nervous symptoms of this disease also improve or entirely disappear as to be convinced that these nervous symptoms are in many instances merely secondary and symptomatic, and in the overwhelming majority of cases the actual clinical picture of colitis membranacea is caused by a severe inflammatory affection of the colon, a malady which originates in the colon itself, or in its immediate vicinity and in some manner, by way of the peritoneum, attacks the colon. Nevertheless cases remain in which only a colica mucosa, therefore a sensory and secretory neurosis of the intestine, is present; however, these cases in my experience do not form the rule.

Colitis muco-membranacea may occur at any age; it is most frequent in middle life, rarer in childhood and in the aged. Women are most subject to the disease, particularly such as suffer from enteroptosis, dysmenorrhea or other sexual ailments; sometimes these develop after abdominal operation, perhaps in consequence of adhesions.

In the periods between the attacks most patients present the symptoms of simple catarrhal colitis with constipation. Periodically these symptoms are intensified by increasing constipation, and culminate in a typical attack of colic, in colitis muco-membranacea, at the acme of which a passage of mucus occurs; this must be regarded as critical inasmuch as subsequently, and particularly after a profuse evacuation of feces, there is rapid and conspicuous improvement, and even relative euphoria for some time.

The colic begins with a feeling of constriction, of burning or rawness in the epigastrium, the left hypochondrium, or the left lumbar region; this gradually increases to severe spasmodic or neuralgic pain of varying intensity which continues for hours or even days, being sometimes accompanied by reflex phenomena of diverse character (spastic constipation). In some cases vomiting occurs during this time; also, although rarely, slight rises in temperature and albuminuria; often we note palpitation of the heart, pulsation of the abdominal aorta, and a rapid, tense pulse. Finally, there is an evacuation of the bowels in which the quantity and composition of the admixed mucus is conspicuous. Often mucus alone is discharged.

The mucus discharged reveals nothing especially characteristic since in simple catarrh of the colon also mucus may be passed in the form of shreds, tubes, globular or grape-like masses. But in colitis muco-membranacea the composition of the mucus is significant; it is hard and tough, much coarser than that usually discharged in intestinal catarrh is membranous, and not dissimilar to croupy membranes although it con tains no fibrin. There is unquestionably no desquamation of the intestinal mucous membrane, as was formerly assumed by some authors. On the contrary, a process of coagulation causes the peculiar change in the mucus. Intestinal epithelia and round cells in great numbers adhere to these mucus shreds. Only when these are absent, and when in the intervals free from attack no signs of intestinal catarrh are apparent, when, therefore, no mucus is discharged, can a purely nervous enteropathy, in the sense of a secretory neurosis or an enteralgia, be considered.

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[1] See the article by Hoppe-Seyler in this volume.

We should never be content with the proof of a catarrh and the presence of accompanying nervous symptoms or sequels of a colitis, for, in most cases, especially the severe ones, the diagnosis is only confirmed when, by a minute study of the conditions and by a clinical examination, perhaps under anesthesia, the cause and the origin of the secondary colitis have been determined.

There are rare cases of colitis muco-membranacea in which the malady terminates with a single attack (Nothnagel); usually, however, the affection is chronic and very tenacious. Many authors believe that colitis muco-membranacea is extremely difficult to cure or even incurable (A. Clark). In my experience the prognosis is no worse than that of other chronic intestinal catarrhs, especially if treated in an institution where by persistent and suitable curative methods excellent results may be attained. It is true there are cases of severe secondary chronic colitis which can be relieved only by internal remedies, and are curable only by gynecological or surgical treatment.

Treatment.—The treatment of chronic intestinal catarrh is designed to regulate the *nutrition* and the *action of the bowels* so that any irritation which may produce or increase inflammation in the intestinal wall is prevented, and an opportunity is given the bowel gradually to recover its tone.

In regard to *diet* it is very difficult to establish general rules, as the peculiarities of each individual case must be borne in mind. The food products which are easily digested, have a high nutritive value, and leave but little residue, are very numerous. We must discriminate from among these, and decide which are best borne by the diseased intestine, which, at the same time, will sufficiently maintain the normal strength, or so improve a reduced or weakened organism that it may again become strong. In many cases, especially such as occur in the debilitated, which run their course with marked irritability of the intestines, it is advisable to begin the treatment with a few days of rest in bed and liquid diet. As soon as possible, however, and while avoiding anything that will demand too great exertion of the digestive organs, I give the previously mentioned normal diet—a mixed diet in which special preference is given to foods cooked with flour—since by the amount and composition of the feces these permit us most easily to recognize deviations from the normal. If, after investigation by a functional test of the stomach and the examination of the feces, disturbances in digestion become apparent, these are combated by the administration of hydrochloric acid, pepsin, pancreatin or pankreon, and papain, and, if these are unsuccessful or insufficient, changes in the diet must be instituted to prevent the undigested and unabsorbed constituents of the food from being propelled into the intestine, where they may produce irritation or processes of fermentation and decomposition, or maintain these. Abnormal decomposition necessitates a limitation of albuminous diet, particularly of meat; abnormal fermentation, a limitation of carbohydrates and foods containing cellulose, and the corresponding substitution for these of other foods.

The irritative forms of chronic colitis—such as run their course with diarrhea or spastic constipation and colic—require a diet which lessens irritation. In the torpid, atonic forms of intestinal catarrh, foods which mechanically or chemically stimulate peristalsis are to be employed, provided that by their use we do not over-irritate the intestinal mucous membrane.

As the type of a diet which will lessen irritation, the *dietary employed in ulcer,* in gastric ulcer, may be mentioned. But it must be remembered that an exclusive milk diet is less well borne in intestinal disease than in gastric disease (some forms of tropical diarrhea, however, form an exception to this rule, and are occasionally cured by a milk diet). Therefore, when milk is not well borne we modify the diet, and give milk diluted with oatmeal gruel or barley water, cooked with fine flour or with one of the numerous infant foods, racahout, hygiama, oatmeal, cacao, etc., or with the many mixtures of flour found in commerce, these to be given in a fluid or pappy form, and then, step by step, we pass to a more abundant diet suited to the individual case; for a time, however, mechanical preparation of the food must be insisted upon (disintegration with a meat chopper).

In diarrhea some foods and fluids enjoy a certain reputation because they have a constipating effect; for example, barley water, rice, cacao, eggs, tea, red wine, whortleberries, and the juice of stewed pears. In the case of tea this is not always true. Neither is it with red wine and veal, and, in the main, not to speak of individual peculiarities, we may say that all foods appear to have a constipating effect which are dissolved and absorbed in the gastrointestinal canal, and therefore leave but little residue.

In contrast with this there are many foods and luxuries which stimulate the intestinal function, increase peristalsis, and even have a laxative effect, naturally varying with the individual. Cold water will stimulate, but much more so carbonated waters, lemonade, beer, grape juice, asti, and champagne. Sugar and drinks containing sugar (sugar water), honey, milk sugar, stewed fruits, marmalade, fruit jellies, etc., produce fermentation in the gastrointestinal canal and by the action of their acids stimulate peristalsis. The effect of buttermilk and of kefyr, of sauerkraut and many fruits, may be referred to their acid effect as well as to the development of gas. Besides the direct action of its acid, with sound fruit the indirect effect of its sugar must be considered. Sour wines and salad have a similar action, and the latter, besides, has the same property as many vegetables and contains numerous undigestible constituents (cellulose) which mechanically irritate the intestine inasmuch as they are not decomposed by bacteria, and thus they distend the intestine. The foods which *mechanically stimulate,* such as the heavy varieties of bread containing bran—bran bread, brown bread, ration bread (brown Tommy), Graham bread, Rademann's DK and Simon's bread, etc.—favorably as these act in atonic conditions of the intestine, are little indicated in the irritable forms of intestinal catarrh; for example, in colitis muco-membranacea and spastic constipation. The same may be said of unripe, unpeeled fruit, of the skins and seeds of grapes and many other fruits, of heavy varieties of cabbage and the legumes when insufficiently boiled.

Food rich in fat (cream, butter, fatty meat, fatty sauces and salads, farinaceous foods cooked in fat) in most persons stimulate intestinal peristalsis; generally, however, with the exception of milk, cream and butter, it is difficult to digest and is a standard for testing the so-called "digestive strength" of the stomach. Coffee, tea, tobacco, and brandy are judged according to their individual effects; the same is true of very salty or spiced delicatessen and the like. The irritation they produce in the intestinal wall varies; gradually, however, the digestive organs become accustomed to them, but subsequently the mucous membrane and musculature of the intestines, the liver, the vascular system, the kidneys and nervous system begin to suffer. Very few luxuries and delicatessen are suitable in dietetic treatment.

Artificial food preparations, I believe, should be used only occasionally, for, as permanent constituents of the dietary, food which is daily prepared afresh in the kitchen is preferable for the healthy as well as for the sick.

The same thought and discrimination should be exercised in regularly supplying the body with food as is devoted to the regular evacuation of the intestinal contents.

In chronic intestinal catarrh accompanied by diarrhea, regular and long-continued intestinal irrigations in the manner described are advisable. Besides lukewarm chamomile infusion or lukewarm saline solution, for this purpose mild mineral waters (Ems water, Carlsbad water diluted with an equal part of warm water) come into question, also dilute solutions of salicylic acid (1:1,000) or very dilute lysol solution (1 or 2:2,000). From solutions of tannin (5:1,000), of ichthyol, silver nitrate (0.5:1,000) and other solutions of silver I have, as a rule, seen no better results than from suitable washings with chamomile water and mineral waters of proper temperature, provided the cause of the diarrhea was not in the lowest portion of the intestine. I have never employed long tubes for intestinal irrigations if the sphincter ani retained its tone. In laceration of the peritoneum alone are we justified in attempting to reach a point above the sphincter tertius, but in deep-seated stenoses we must try to pass the stenosed area by means of long, soft, rectal tubes.

These watery enemata may also be employed in chronic intestinal catarrh which runs its course with constipation and retention of feces. There is no disadvantage from their continued use provided the quantity of fluid is no larger than is necessary to produce evacuating peristalsis. But the frequent, or even regular, employment of so-called high intestinal injections of 2, 3 or more liters is not justifiable, and I have repeatedly heard patients state that their intestinal disorders, particularly of the atonic form, were aggravated by these forced injections. On the other hand, it cannot be denied that large injections into the intestine, if well borne, are particularly beneficial in cases of secondary colitis in which old peritoneal adhesions hinder the passage of feces. Inflammatory processes in the pelvis, even those which have run their course, in some women prevent the employment of even small injections on account of the pain they produce; that is, the quantity of water which can be injected into the intestine without causing pain is insufficient to stimulate an active peristalsis; the same is true in many cases of spastic constipation.

The most effective and, at the same time, the mildest treatment is by *oil enemata,* which Kussmaul and I employed in all affections of the colon running their course with constipation. Ten years ago I described their use, technic and physiologic effects (*Berliner klin. Wochenschr.,* 1893, Nr. 3 u. 4), and since that time the method has been extensively and most successfully employed in hospital and private practice.

The oil, lukewarm olive oil, poppy-seed oil, or sesame oil, if of proper temperature and carefully injected, penetrates high up into the intestine, similarly to the oil in the wick of a lamp. The passage of the oil is favored by placing the patient in a proper posture—usually the left lateral position or the dorsal decubitus with raised pelvis, under some circumstances even the knee-elbow position—by the pendular movement of th intestines, and by the physical composition of the oil (absorption).

Primarily the oil lessens the irritability in the intestine, perhaps actin like ointment upon a wound: It coats the intestinal mucous membran and protects it from pressure and friction, loosens from it the mucus an adherent feces, limits the absorption of fecal fluid in the dissolved prod ucts of decomposition at the oiled areas of the intestinal wall, and thereb prevents a marked inspissation of the feces.

There is no special difficulty in retaining the oil in the intestine f hours. While so retained, free oleic acid is split off by the action bacteria, by the intestinal juices higher up in the intestine, and also by other undecomposed digestive juices, particularly by bile, and this causes a marked increase in the activity of the acid contained in the oil (0.34–3.97 per cent.). The oil which has become acid (simultaneously with the split-off glycerin, the soaps formed from the oleic acid, and the alkali of the intestinal juices) stimulates the bowel in a manner closely corresponding to intestinal peristalsis, and as soon as it reaches the necessary degree of acidity for a chemo-reflex causes an evacuation. It also acts upon the excretion of bile and intestinal fluid, the oleic acid split off having a reflex action. Only a small portion of oleic acid is saponified and absorbed, for at least as much oil is evacuated as was injected into the intestine.

The exceedingly mild effect of the oil may be explained by the theory that the oil in the intestine acts only as an evacuant after it has loosened the fecal masses from the intestinal wall and has rendered them pliable and smooth, so that a comparatively slight peristaltic action is sufficient for their expulsion. The undecomposed oil present in excess produces no further irritation of the mucous membrane, at least patients rarely complain of any such subsequent effects from the oil as follow the taking of laxatives; on the contrary, the evacuations, as a rule, promote their comfort. Diarrhea rarely occurs after enemata of oil, although, when the effect of the oil is at its height, evacuations may occasionally take place several times during the day.

When the decomposed oil and the feces have been expelled from the colon by one or several evacuations, the previously described chemism again affects the retained oil which adheres to the mucous membrane of the bowel. If the quantity of oil is small, a long time will be necessary to stimulate peristalsis and to form oleic acid, glycerin and quantities of soap. The number of evacuations will, however, become less until, finally, an additional amount of oil will be necessary to regulate the bowels.

According to this view, and wherever indicated, a treatment with oil may be instituted which will meet the individual circumstances of the case. This indication is found in *all forms of constipation,* particularly in such *as run their course with irritative symptoms,* but especially in inflammatory, catarrhal and ulcerative processes of the colon immaterial whether these are accompanied by peritoneal—non-perforating—symptoms or not.[1]

The *dose of oil* employed in the enemata depends upon the size of the patient and the seat of the pathologic focus in the intestine. For adults the first injection should contain from 200 to 400 grams, for children 50 to 100 grams of oil at the temperature of the body; this is stimulating effect upon peristalsis. True spa treatment often fails to cure the severe forms of chronic colitis. I have, however, known great benefit to follow the treatment at Kissingen, Homburg, Tarasp, Carlsbad, Wiesbaden, and other resorts, provided the use of the waters was instituted after the most acute disturbances had been relieved by treatment in a sanatorium or at home, especially by the oil cure.

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[1] Other indications and individual points are given in the original article, quoted above, in the *Berliner klin. Wochenschr.*, 1893, Nr. 3 u. 4.

In the majority of cases of chronic intestinal catarrh of gastric origin, also in the numerous secondary complications which involve the stomach, systematic local treatment of this organ by lavage (early in the morning upon an empty stomach) is very advantageous. The effect of lavage upon the appetite, upon the functions of the stomach, upon the upper small intestine, upon the liver, upon the blood and the kidneys, and last, but not least, upon the morale of the patient is so well known that I need not enter into details; in diarrhea alone does gastric lavage occasionally have an unfavorable effect, and in these cases it should never be employed.

A number of other remedies indirectly influence the pathologic processes in the intestine and the disturbances in function dependent upon these. Heat, for example, is beneficial by lessening irritation (warmth of the bed and rest in bed); hot dry cloths, hot bottles, thermophores, moist heat by means of compresses (cataplasms) are all useful for relieving abdominal pain and colic. As a rule, patients with disease of the intestine and particularly those who suffer from diarrhea and whose nutrition has been damaged require a great amount of heat. They are easily chilled; when the physician advises them to wear an abdominal binder they follow the advice with excessive eagerness. In the winter there is no objection to wearing a flannel binder, but usually the wearing of these bandages does harm rather than good, since they weaken the skin which steadily becomes more susceptible to the effect of cold.

Cold applied to the skin reflexly stimulates peristalsis. Its hydrotherapeutic employment in cases of catarrh combined with intestinal atony produces remarkably good results; for this purpose Sitz baths of brief duration, bathing of the abdomen, friction, half-baths, douches, river and sea baths, may all be employed to relieve constipation. Besides, by improving the circulation, these procedures have a curative effect upon the intestinal catarrh which may even be increased by the addition of salt to the bath; therefore, salt baths, mineral baths and peat baths are advisable.

In suitable cases *electricity* may also be employed: The anode of the galvanic current for the amelioration of pain which originates from spastic conditions and neuralgia; faradization of the abdominal wall to stimulate intestinal movements.

Massage is indicated in some of the cases. When there are irritative conditions in the intestine and the peritoneum, pains, spastic constipation and the presence of hard fecal masses in catarrhally inflamed portions o the intestine, massage is contraindicated, while in the atonic and torpid conditions which mark some cases of chronic colitis brilliant results are sometimes attained by massage. Vibration massage is usually less harmful than manual, and is often well borne when the latter is contraindicated. We cannot, however, be certain of its advisability, for apparently, like many of these procedures, it presupposes more or less psychical susceptibility.

Medico-mechanical treatment and gymnastic exercises combined with spa and bath cures are very useful for the constipation of chronic colitis.

Besides these auxiliary methods of treatment, in the severe forms of secondary colitis (colitis muco-membranacea) which originate from inflammatory processes in the pelvis and abdominal organs, gynecological measures must be considered: Vaginal treatment, rectal treatment by bougies, under some circumstances even laparotomy for the purpose of breaking up peritoneal adhesions or the extirpation of tumors or diseased organs, for example, of an ovary, of the uterus, or of the tubes. I am not yet ready to relate my experiences with thiosinamin injections in cases of peritoneal adhesions which developed after abdominal operations, but the subject is a promising one.

III. INTESTINAL TUBERCULOSIS

Tuberculous infection of the intestines is caused by tubercle bacilli which reach the mouth in some manner and are swallowed. Frequently the infection is self-conveyed by the sputum of a tuberculous individual, who swallows it and thus introduces numerous tubercle bacilli into the intestine; but the tuberculous sputum of others may be directly thrown into the mouth of a healthy person by coughing, or it may be conveyed from the lips of a patient to a healthy person in the form of the finest particles. Dried tuberculous sputum in the air contains bacilli which upon inspiration do not all reach the deep respiratory passages and the lungs, but may adhere to the wall of the buccal cavity and be swallowed with the saliva. Little children playing with earth frequently introduce tubercle bacilli into the mouth with their dirty fingers as well as with toys and other articles which they lick or bite. From tuberculous wounds, with soiled fingers, instruments or any other object, children as well as adults may introduce the tuberculous poison into the mouth.

A mode of infection the importance of which was for a long time undervalued, and subsequently for a time exaggerated, is by means of food and drink containing bacilli (feeding tuberculosis). Since the discovery of the tubercle bacillus by Robert Koch, bacteriologic investigation has revealed a number of microörganisms resembling tubercle which at first were considered to be different in nature but are now known to be a more or less *common variety of one fundamental form*; they have developed by ably in the direction of the vascular division, therefore vertically to the longitudinal axis of the intestine; thus the annular or girdle-like ulcers develop.

The borders of the tuberculous ulcers are usually infiltrated, the margins are elevated, and often undermined and permeated by the characteristic and more or less caseous and decomposed nodules. The latter are found also upon the base of the ulcer, which may be of varying depth, the floor being formed by the submucosa, the muscularis, and even by the serosa. At the autopsy we invariably recognize the seat of tuberculous intestinal ulcers even from without by the redness and thickening of the serosa and its permeation by small nodules. This circumscribed tuberculous peritonitis commonly favors the formation of adhesions and the extensive agglutination of diseased portions of the intestine with other organs covered by the peritoneum and the abdominal wall, and renders the perforation of tuberculous intestinal ulcers a relatively rare occurrence; when it does occur it is less dangerous because the succeeding peritonitis is sacculated. Where there is no serous covering of the intestine, as in the rectum, ulcerative tuberculosis leads to perirectal abscesses and subsequently to internal, external, and complete anal fistula.

Tuberculous granulation proliferation may terminate in caseation and ulcerous decomposition as well as in induration, according to the action of the bacterial toxins and the composition of the base on which they develop, and also perhaps the constitution of the patient (Ziegler). Hence we find in one case extensive and advancing ulceration in the intestine, in another, on the contrary, a conspicuous tendency to cicatrization and induration by connective tissue proliferation. Undoubtedly the cicatrization of a tuberculous ulcer must be regarded as a favorable process and indicative of a cure. This healing tendency may, however, in one sense be a disadvantage to the organism, for, as pyloric stenosis may develop in consequence of the cicatrization of a gastric ulcer at the pylorus, so by the cicatrization of an annular tuberculous ulcer of the intestine *constriction of the intestine* and severe disturbances in function may be brought about. Such cicatricial stenoses in the intestine occur simultaneously with tuberculous ulcers; I have repeatedly seen them multiple in the same case.

The most extreme constrictions of the intestine are the ring-shaped, cicatricial stenoses which originate from annular ulcers, and are very often short. The cicatrices which project spur-like into the lumen of the intestine and encompass only a portion of its circumference, at least in the small intestine, do not decidedly affect the permeability of the latter. Nikoljski draws the contrast between tuberculous and fibrinous tuberculous stricture which does not originate from an ulcer—consequently does not permit the recognition of cicatricial formation upon the mucous membrane—but arises from simple connective tissue proliferation in consequence of tuberculous infection of the intestine—peribasillary sclerosis. A third form of tuberculous intestinal stenosis is the so-called hypertrophic stricture which is characterized by great thickening of the intestinal wall, a thickening which involves all of the layers, the mucous membrane by polypus proliferation, the submucosa and muscularis by hypertrophy, and the serosa by a decided connective tissue development which, by its permeation with masses of fat, increases the resemblance to a tumor. The differential diagnosis between carcinoma and tuberculosis may be perplexing even after extirpation of the stenosing tumor, until the microscope decides the question—as in one of my cases. In this hypertrophic form of tuberculous intestinal stenosis the submucosa with its vessels is most markedly implicated; some authors consider hypertrophic intestinal tuberculosis as of hematogenous origin (Nikoljski).

SYMPTOMS

The *clinical phenomena of intestinal tuberculosis* are manifold, and in the advanced stages very peculiar.

The initial stage of the affection almost always eludes observation, although it cannot be assumed that the inflammatory processes succeeding tuberculous infection of the intestine run their course without creating functional disturbances (pre-tuberculous diarrhea?).

From *indistinct, dyspeptic, intestinal disturbances* which in children, and not rarely in adults, are accompanied by a febrile movement, in the course of time, often very slowly, well developed clinical pictures are formed which correspond to the advancing pathologic changes in the intestinal wall and the mesenteric lymph-glands; these, in many cases, bear a relation to the symptom-complex of chronic intestinal catarrh with those anomalies in regard to the bowel movements which have been described. As a rule, however, just as in the pathological processes in the intestinal wall, ulcerative symptoms predominate. In every case peritoneal irritative phenomena may be superadded, either transitorily or permanently.

The symptoms of *subacute* and *chronic intestinal catarrh*, especially the increased excretion of mucus, may be due to the inflammatory process in the intestinal wall. Often, however, in compact as in diarrheic intestinal discharges, mucus is absent, while the intensity of other irritative symptoms does not permit us to assume the cessation of the inflammatory processes. Periodically mucus in greater masses is often admixed with the feces. In such cases the increased excretion of mucus is not always the sign of an exacerbating tuberculous inflammation, for functional disturbances accidentally produced by a long-existing ulcer, above all spastic contracture and fecal retention, may often produce in the mucous membrane of the affected intestinal area a great excretion of mucus, an irritative condition resembling colica mucosa.

In their nature and intensity the *ulcerative symptoms* of intestinal tuberculosis depend much less on the extent of surface and the depth of the tuberculous ulcerative process than on the intensity of the irritation of the exposed intestinal nerves upon the floor of the ulcer. If these are absent in ulcerated areas, it may happen that numerous and comparatively large tuberculous ulcers in the small intestine as well as in the colon run their course entirely latent, often causing no functional disturbance, while in other cases an isolated ulcer in the colon, if this implicate an important nerve branch, may produce the most severe irritative symptoms. The latter may be of a motor, sensory or secretory nature; to these must be added, as other symptoms of ulcer, hemorrhages and peritoneal and perirectal inflammations.

The motor irritative phenomena are most frequent in intestinal tuberculosis. *Diarrhea* is, therefore, the most common symptom of intestinal tuberculosis, and, when persistent, the possibility of intestinal tuberculosis must always be considered unless other symptoms indicate a different affection. In cases of tuberculous intestinal ulcers we now and then see typical spastic constipation combined with the previously mentioned painful evacuations of mucus which resemble mucous colic. The distress is often localized in the ileo-cecal region, and at first is usually attributed to an acute affection of the appendix. Violent tenesmus, often combined with the passage of blood, is produced by tuberculous ulcers of the rectum. In some cases the irritation of the rectum, and even that of the sigmoid flexure, simulates the picture of nervous hyperesthesia of the intestine. Small masses of dejecta which reach the rectum produce an irresistible desire to relieve the bowels, and this is frequently repeated at short intervals to the great distress of the patient. Since in these cases the evacuations are very small in amount, not rarely there is a paradoxical retention of feces, a constipation-diarrhea.

Peristaltic unrest accompanied by loud intestinal rumblings is an almost invariable symptom in the chronic diarrhea of intestinal tuberculosis; if, however, it continues after the diarrhea ceases, or is more marked during the periods of constipation, this increased peristaltic unrest is often the first sign of a beginning stenosis of the intestine. Added to it in the course of weeks and months are peristaltic pains which occur at more or less regular intervals, often every few minutes, with a characteristic tension of that portion of the intestine anterior to the obstruction. I shall later discuss this symptom of intestinal constriction.

The diarrhea of intestinal tuberculosis is frequently associated with pain; and the previously mentioned spasmodic state of the intestine, the contracture as well as the over-distention sometimes intercurrent with the former but also occurring without it, is often marked by excessive pain. During or before defecation, when hard feces pass through the ulcerated intestinal area, there is often true *ulcer pain* of great severity in the region of the colon, especially in the lower portion. In such cases every evacuation distresses the patient for more or less time, often only for a few minutes,

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logic processes which favor the healing of ulcers may, however, have a decidedly injurious effect upon the permeability of the intestine. Cicatrization of an ulcer leads to cicatricial stenosis; diffuse infiltration and

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Pain confined to definite areas of the abdomen, persisting for days but varying in intensity, and not markedly influenced by the intestinal function, may be regarded as of *peritoneal origin*, especially when there is local tenderness on pressure and a rise in temperature, and when nausea and a tendency to vomit are accompaniments.

These peritoneal symptoms may be acute and of great severity, and may be the forerunners of the gravest form of peritonitis, that due to perforation. But in intestinal tuberculosis an insidious onset and a course showing complications and frequent relapses is more common. Often the pain and the local sensitiveness to pressure are the only symptoms of the condition; sometimes a circumscribed, plastic, fibrinous exudate is formed, the demonstration of which may be impossible; after these peritoneal attacks adhesions of the intestine invariably form, and not infrequently cause new functional disturbances of the intestine. These are slightest in the very common serous exudates, often of enormous extent, which occur in tuberculous inflammations of the peritoneum.

Hemorrhage from the intestine is the most positive sign of ulcer. Tuberculous intestinal ulcers, however, rarely show a tendency to bleed, for the tubercle, as such, is non-vascular, and, furthermore, it is a peculiarity of the tubercular toxin speedily to obliterate small vessels. Nevertheless, in intestinal tuberculosis of considerable intensity and very difficult to control I have several times seen hemorrhage, particularly from the lower portions of the bowel. In such copious hemorrhages mechanical injury to the vessels from impacted fecal masses probably plays a greater rôle than erosion of the wall of the vessel—similar to the condition in pulmonary cavities—by a rapidly advancing process of ulceration. Yet in no other pathologic process is there such great recuperative power, such a disproportion between destruction and restitution, as is peculiar to the tubercular; consequently in severe and repeated hemorrhages from tuberculous intestinal ulcers we must never give up hope. I have seen the hemorrhage arrested, and even amelioration for years of all the severe anemic conditions which followed such hemorrhage.

The transformation of tuberculous granulation tissue into cicatricial connective tissue, connective tissue proliferation in the surroundings of tuberculous ulcers, and the formation from the plastic exudate of pseudomembranes and indurations upon the serosa of the intestine, corresponding to the seat and the areas surrounding the tuberculous ulcers, in some cases assist the conspicuous tendency of the latter to heal. These pathologic processes which favor the healing of ulcers may, however, have a decidedly injurious effect upon the permeability of the intestine. Cicatrization of an ulcer leads to cicatricial stenosis; diffuse infiltration and thickening of the walls of a portion of the intestine prevent the propulsion of the peristaltic wave. Induration of the serosa of a section of the bowel also interrupts peristalsis, and pseudo-membranous adhesions readily produce traction and kinking in the lumen of the intestine, and consequent constriction and deformity which render the propulsion of the intestinal contents difficult.

By compensatory hypertrophy of the intestinal musculature above the point of obstruction these conditions may for a long time remain dormant and disturbances of fecal movement be compensated for. The diffuse, rigid infiltration and indurative thickening of definite sections of the bowel first become noticeable because these assume the form of a cylindrical strand or of a tumor of irregular shape and extent. These may then be palpated, but only in areas of the abdomen where the intestine and particularly the colon are readily palpable under normal circumstances, i. e., in the region of the cecum and of the sigmoid flexure. Often the beginning of the colon, the cecum with the appendix, and the ascending colon, are found to be the seat of the tuberculous infection. A tumor-like mass is felt in the right iliac fossa, and the sigmoid flexure in the left iliac fossa is found to be thickened, whereas, in many cases, these pathologic changes in the flexure of the colon, particularly upon the left side, cannot be determined during life.

In the small intestine, on account of the fluid or thin, semi-solid consistence of the intestinal contents, disturbances in compensation appear only late, after the intestinal constriction has reached an extreme degree. The same is true of the colon, particularly in the cases which run their course with chronic diarrhea. As, however, in most cases only a portion of the ulcer is cicatrized, while another is still ulcerated and irritated by the intestinal contents, every spasm due to the irritation of the ulcer, any accidental inspissation of the feces, may disturb compensation in stenosis of the colon.[1]

The first sign of a disturbance in compensation is the distention of the intestine above the stenosis. It may be circumscribed and soon disappear, or may rapidly increase in extent and distribute itself over the entire abdomen. Great peristaltic unrest combined with gurgling and rumbling in the abdomen then follows. With the increase of the abdominal distention the diaphragm is pressed upward, rendering respiration difficult and causing great discomfort. The distress is increased when nausea, eructations, singultus and a tendency to vomit appear, or when the accumulations of decomposed chyme from the small intestine regurgitate into the stomach and the eructations of gas or the vomitus acquire a disagreeable odor.

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[1] In a tuberculous stenosis of the ileo-cecal valve I saw a chronic invagination of the diseased lower ileum into the cecum and ascending colon. The replacement was comparatively easy, and subsequent invagination was prevented by a suitable bandage. Death finally occurred, not from intestinal occlusion, but chiefly in consequence of a complicating valvular lesion.

From time to time marked peristaltic waves occur in the greatly distended abdomen; and these may be due to extreme mechanical distention of the intestine, to chemical irritation from the highly decomposed intestinal contents, to reflexes, to the overloading of the stomach in eating or drinking, to cooling of the abdominal surface when the clothes are removed, or to mechanical irritation by percussion or palpation during the examination of the abdomen, particularly, however, from friction of the belly; these waves forcibly propel the fluid intestinal contents downward toward the obstruction, and so tensely fill a greater or less number of intestinal coils in front of the stenosis that their contours may be distinctly seen through the thin abdominal wall. These inflated intestinal coils are stiff upon palpation—*intestinal rigidity*—those nearest the stenosis often being as hard as wood, tumor-like. With this violent peristalsis agonizing pain, resembling uterine pain, sets in and gradually increases to the highest intensity. This intestinal rigidity and pain remain at their acme for a few seconds, occasionally for a minute, but rarely longer, and then suddenly cease, while the fluid contents, accompanied by loud rumbling and gurgling and apparently under the contraction of the overdistended intestinal coils (therefore antiperistaltically), are again forced back. Before this regurgitation occurs we occasionally hear in the stenosed intestinal area a murmur of long or short duration, a sort of squirting sound, which is due to the forced passage of the fluid and gas-containing intestinal contents through the stenosis.

During a peristaltic wave of this kind the pain is often so great that the face of the patient becomes contorted as in severe colic; the eyes sink in their orbits, the nose becomes pointed, and the expression of anxiety and suffering thus presented resembles the facies hippocratica. Cold sweat often appears, the pulse becomes small and weak, even intermittent; in very debilitated persons syncope follows.

Gradually the patient becomes so accustomed to this wave-like peristalsis that it no longer causes pain. This is not a good omen, for in such cases it certainly indicates that the tonus of the intestine has been impaired, that a great dilatation of that portion of the bowel situated in front of the obstruction has taken place, or there is regurgitation into the small intestine. Improvement in such cases is only apparent and transitory, and symptoms of intestinal occlusion rapidly appear.

In both varieties of intestinal tuberculosis, primary and secondary, the *local* symptoms which have been described are the same, that is, they *may* be the same. The *general clinical picture*, however, varies extraordinarily, and in secondary intestinal tuberculosis is influenced to a great extent by the tubercular infection of other organs, particularly of the lungs, to which intestinal tuberculosis is superadded.

tenaciously to the bed-clothes and the body linen of the patient, and by the experienced physician is easily recognizable upon entering the sick room. Besides the functional weakness of the digestive juices and the limitation of intestinal absorption in consequence of increased peristalsis, a cause of this immoderate intestinal decomposition is the profuse admixture of pathological secretions from the intestine with the feces. Fat absorption suffers early and most severely in the chronic diarrhea of patients with intestinal tuberculosis, and to an extraordinary degree in those cases of primary or even of secondary intestinal tuberculosis in which the mesenteric lymph-glands are to a great extent diseased. Nevertheless, there is less fat in diarrheic feces than is found in steatorrhea with disease of the liver and of the pancreas.

Corresponding to this excessive intestinal decomposition we frequently find in chronic diarrhea from intestinal tuberculosis a decided increase of indican and of ethyl sulphuric acid ($C_2H_5HSO_4$) in the urine.

TREATMENT

The treatment of intestinal tuberculosis is exceedingly difficult, especially when it appears as a complication of tuberculous disease of other organs, and the obstinate diarrhea which accompanies it makes impossible the employment of one of the most important remedies in the treatment of tuberculosis, namely, hypernutrition. Recognizing this condition in pulmonary tuberculosis, we must be exceedingly careful to see that the sputum rich in bacilli does not find its way into the intestine by deglutition. Unfortunately, we cannot prevent small children from swallowing sputum; even with older ones it is difficult to enforce this prophylactic measure, as well as with very sensitive patients who regard the expectoration of sputum as a violation of estheticism which is more flagrant than their violation of the laws of hygiene by swallowing it. The educational effect of sanatoria for pulmonary diseases is especially evident in their recognition of the preventability of autoinfection.

In the *prophylaxis* of primary intestinal tuberculosis, certain sanitary laws in regard to the inspection of meat and the consumption of meat from animals infected by tuberculosis are operative. But a greater danger than confronts adults by the use of meat threatens nurslings and young children by the ingestion of milk from tuberculous cows. Maragliano and other investigators made an attempt to immunize these animals and to obtain from them a curative serum. v. Behring systematically carried out his immunization theory which he designates as Jennerization not only in a few animals but also in entire herds of cattle. According to his experience, it seems to be possible to introduce antibodies into nurslings and small children by ordinary nutrition with the milk of cows that have been rendered immune to tuberculosis, and thus to tide them over the dangerous period of contamination.

Many attempts have been made to find a specific curative serum for tuberculosis. All hopes based upon remedies new or old, or upon patent medicines, have been blasted, and the enormous amount of energy and time consumed to find a specifically acting serum has led to no satisfactory results. Preventive inoculations with attenuated tuberculous virus and injections into the tuberculously diseased body of other bacteria antagonistic to tubercle bacilli have not found their way into practice.

In the year 1890, at the International Medical Congress in Berlin, the communication of R. Koch that he had obtained a product from tubercle bacilli which would have a specific effect upon the tuberculously diseased organism, this substance being first designated as Koch's *lymph* and later as *tuberculin*, made a great furore, and awakened intense enthusiasm; but here popular enthusiasm was rapidly followed by a depressing disillusionment which lasted until, at the beginning of this century and after years of critical labor and study, it was demonstrated that Robert Koch's remedy forms the basis of an etiologic specific therapy of tuberculosis, and that among numerous tuberculosis specifics which had in the meantime been produced his tuberculin deserves preference as a diagnostic remedy.

From his researches R. Koch assumed that *incipient phthisis* could certainly be cured by his remedy; "this may still be true of the cases not too far advanced." It is therefore of the utmost importance for the success of the specific treatment that we use Koch's tuberculin *as early as possible*. In doubtful cases which appear to be tuberculosis we now employ the tuberculin injections in order to make the diagnosis certain; hence, those cases are particularly suitable for tuberculin treatment in which a positive reaction follows the diagnostic injection. R. Koch has produced various tuberculin substances; but only two of these are employed, the old tuberculin, the original Koch's lymph, and the new tuberculin which is an emulsion of tubercle bacilli. For diagnostic purposes the former is preferable; but for therapeutic purposes after the old tuberculin has given a positive reaction in a diagnostic test, and when this therapeutic use does not produce the desired effect, the new tuberculin should also be tried.

The technic is simple and presupposes strict asepsis; prior to the diagnostic injection, the temperature of the patient should be determined by a temperature record taken every three hours for two or three days. The Höchst tuberculin is used in a pipette divided into a hundred parts; it is diluted with boiled water in a proportion of 1:1,000 or 0.1:100, so that each division of a Pravaz syringe will then contain .1 of a milligram of tuberculin. This dose is injected into the back between the shoulder blade and the vertebral column, and the temperature is taken early in the morning as there is usually a reaction after twelve hours. "A rise in temperature of .9° F. above the normal temperature is regarded as a reaction. If with .1 of a milligram there is no reaction but a rise in temperature of about .5° to .7° F., in the next injection, which should follow in about three days, a dose of .2 of a milligram will produce a distinct reaction. If, after the first injection, there is no rise in temperature, for the second injection I give .5 of a milligram, for the third, one milligram, etc., until in adults 10 milligrams at most are given; in children, according to Koch, 5 milligrams must be regarded as a maximum dose. If these doses fail to produce a rise in temperature of at least .9° F. the reaction may be regarded as negative" (Moeller).[1]

The therapeutic employment of tuberculin is the same as the diagnostic. The dose is regulated by the reaction in a concrete case, a marked individual reaction is avoided, and the injections should not too rapidly succeed each other, being usually given not oftener than twice a week. A decrease in the intensity of the reaction after large individual doses of tuberculin indicates improvement. Recovery may be assumed if, after months or years, repeated diagnostic injections of tuberculin no longer produce a reaction.

In addition to the specific treatment of tuberculosis, *symptomatic treatment* of the bowel disturbances in intestinal tuberculosis should not be left out of consideration. In far advanced cases which are unsuitable for specific treatment, or in which there are complications, the greatest weight is to be attached to the symptomatic treatment of the intestinal affection, and this must be pursued according to the plan proposed in the previous chapters.

Therefore in the chronic diarrhea of intestinal tuberculosis flushing of the intestine from above by drinking mild mineral waters as warm as possible or infusions of peppermint and other aromatic drugs early in the morning on an empty stomach, or the persistent irrigation of the intestine from below with dilute saline solutions, mineral waters (Ems, Selters, Carlsbad, Vichy), or chamomile tea may be resorted to, possibly with disinfecting or deodorizing substances, such as salicylic acid (1:1,000), creosote, and perhaps menthol. When there is immoderate intestinal decomposition menthol in the form of a coated pill may be given frequently and at regular intervals during the day until the odor is no longer perceptible. Thymol, which markedly corrodes the mucous membrane of the mouth, must be given in gelatin capsules, or, like salol and iodoform, in Sahli's glutoid capsules. In conclusion, small doses of opium which should be invariably given at meal-time (in pill form in combination with hydrochloric acid or extr. ligni campech.) are worthy of trial, as also the occasional administration of bismuth and tannic acid preparations and alum whey.

When there is constipation, either occasional or persistent, laxatives by mouth are to be avoided, as these are liable to damage the general nutrition by wasting unabsorbed nutritive material, and the bowels must be regulated by systematic oil treatment. Occasionally, instead of pure poppy-seed oil or sesame oil, the addition of creosote to the oil ($\frac{1}{2}$ to 1 per cent.) appears to be beneficial. In inflammatory and ulcerative processes in the colon and in the rectum, oil enemata of proper proportions, regularly given at a suitable time, render excellent service. They exert a powerful effect upon the motor and sensory irritative phenomena, and favor and promote the healing of wounds by the protection which they afford to the exposed and granulating tissues. When there is marked tenesmus, an addition of tincture of opium (5 to 10 drops) to an oil enema is useful, provided the opium does not lead to constipation and the inspissation of the feces.

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[1] See chapter by Moeller upon "The Specific Treatment of Chronic Pulmonary Tuberculosis" in the "Handbuch der Therapie," etc., by Schroeder and Blumenfeld. Barth's Verlag, Leipzig, 1904.

With the careful observance of all the conditions which may arise, we undoubtedly have in the oil treatment a prophylactic as well as curative measure for hemorrhages and intestinal ulcers, at least the deep-seated ones, and suited to the individual requirements. By the addition of bismuth subnitrate to the oil (rubbing the bismuth with lukewarm oil in a mortar), I believe that in many cases I have promoted and hastened the healing of ulcers in the rectum and the lower colon. The effect of the bismuth and oil upon hemorrhages did not appear to be any greater than that of pure oil.

On the other hand, bismuth is a valuable remedy, if applied in substance directly to the ulcerated granulating surfaces in the rectum. This can be accomplished in the following manner: After introducing a cylindrical intestinal speculum as high as possible (and removing the obturator) the intestine is cleansed by irrigation; then by means of an insufflator, or by a glass spoon with a long handle, or by an applicator which can be introduced through the intestinal speculum, the latter being then slowly withdrawn, bismuth can be applied to the diseased intestinal mucous membrane. Touching the ulcerated surface with a 1, 2 or 3 per cent. silver nitrate solution prior to the application of bismuth has often been found beneficial, but the silver nitrate frequently so stimulated peristalsis that the bismuth was soon ejected. The same is true of liquor ferri sesquichlorati applied to bleeding ulcers and of tampons in the rectum. For this reason I have, as a rule, used locally either oil or bismuth alone, or bismuth suspended in water as in the treatment of gastric ulcer. This was injected into the intestine, and if hemorrhage from the bowel followed I administered alum whey to the patient.

In a few instances adrenalin was employed as an addition to small enemata of oil (20 to 30 drops of a 1–1,000 alcoholic solution) or as an addition to injections of water for rectal ulcers which showed a tendency to hemorrhage; I have as yet come to no conclusion regarding the value of this remedy.

In cases of ulcerating intestinal tuberculosis with profuse diarrhea and an admixture of blood in the diarrheic feces, besides such measures as are necessary to arrest the diarrhea I have administered gelatin by mouth and have also given it in small enemata. Bismuth continues much longer in suspension in gelatin than in water, therefore gelatin and bismuth enemata are sometimes very effective in the local treatment of ulcers. Finally, when tuberculous ulcers in the rectum may be reached, cauterization (under anesthesia) with a Paquelin cautery may be employed to quiet local hemorrhage. I have known this method of treatment for hemorrhages to be very beneficial. Cauterization is unsuitable for the general treatment of rectal ulcers because a single scarification is insufficient, and repetitions of the process, which are only possible under anesthesia, are too painful, the pain often lasting for several days afterward.

In the *surgical treatment* of intestinal tuberculosis many measures may be indicated, but these vary according to the individual circumstances of the case. In stenosis of the intestine in which a tuberculous process so lessens its permeability that a condition of motor insufficiency develops, the obstruction must be relieved by surgical aid or removed. In some cases the mode of operation can only be decided upon after the abdominal cavity is opened and we have recognized the exact anatomical relations by palpation with a finger or by inspection. If, in a given case of intestinal stenosis, the pathologic focus shows local limitation, resection of the stenosed intestinal area must be attempted, and if in another instance the pathologic changes show great local extension, enteroanastomosis must be performed to overcome the narrowed or occluded intestinal coils. In cases in which none of these methods of operation can be carried out because of technical difficulty, or on account of advancing debility of the patient, we must be content with enterostomy at a suitable point. In some cases of tuberculous intestinal occlusion the formation of such an artificial anus is an *indicatio vitalis.* The arrest of the difficulties, and the marked improvement in local and general conditions after this comparatively slight symptomatic operation justifies its performance also in cases of intestinal tuberculosis in which there is no organic stenosis, but in which, however, extensive ulcerative processes in the colon cause profound subjective disturbances, such as severe diarrhea which cannot be controlled by internal remedies, and especially hemorrhage. Colostomy above the diseased intestinal region excludes the latter from taking part in the labor of digestion, induces much better conditions for the healing of the ulcer, renders possible a more active local treatment of the diseased intestinal mucous membrane, and, what is more important, enables us to give sufficient nourishment and a general treatment. Whether the artificial anus must later be closed depends upon the further progress of the case.

Tuberculosis of the rectum most frequently necessitates surgical measures because inflammatory and ulcerative processes in this portion of the intestine which is not covered by peritoneum readily penetrate from the intestinal wall to the perirectal cellular tissue, or perforate it and there form abscesses which must be incised. Both incised and spontaneously perforating perirectal abscesses leave the familiar internal or external anal fistula which may also be complete, for the treatment of which extensive division through the sphincter is the best remedy.

The tuberculous patient in all stages of his disease needs every possible advantage that can accrue from a hygienic mode of life and careful nursing. Patients with intestinal stenosis do not reap the same benefit from treatment in sanatoria and large institutions for the cure of tuberculosis as those suffering from pulmonary tuberculosis, since, owing to the nature of the intestinal disturbance, their nutrition necessitates greater specialization than is practicable in a large institution in which general over-nutrition is to be carried on for some time. Nor are they adapted for treatment in high altitudes and at the seashore, for they are often very susceptible to changes of temperature; hence bath cures, salt baths, and (when intestinal tuberculosis is combined with peritonitis) peat baths can only come into consideration after the intestinal disturbance shows a certain degree of improvement or has become latent.

Until such amelioration occurs, *nutritive therapy* must be one of the chief aims of the treatment, and for this the fundamental laws have been detailed in preceding chapters. As soon as the condition of the intestine will warrant it we order a mixed diet, preferably of boiled flour foods, in such quantities as may substitute for losses which have taken place in consequence of fever, diarrhea and the under-nutrition due to the latter, and will finally bring about an increase in weight. Care and observation by coproscopy will determine the proper additions of fat to the nutrition, for it has been proven by experience that in intestinal dyspepsia due to tuberculosis fat absorption suffers early and continuously, and in such a way that even the most perfect of foods, milk, is often not well borne.

CONSTIPATION AND HEMORRHOIDS

By J. BOAS, Berlin

Constipation and hemorrhoids, as is well known, belong to the most common affections of the intestinal canal. Perhaps because scientifically resultless, some physicians regard these conditions as a *quantité negligeable* —for there is in medicine a certain unmistakable timocratic principle. Possibly, even to-day, the opinion is extant that constipation and hemorrhoids are slight, though troublesome, evils, and, here and there, an impression exists that internal medicine taken altogether is powerless permanently to relieve this condition.

I hope to convince all that neither of these views is correct. Above all, I wish to state explicitly that the treatment of constipation and hemorrhoids is one of the most fruitful fields of internal medicine, and that it is our imperative duty as physicians thoroughly to acquaint ourselves with this condition since it represents the principal branch of the so-called "Nature Cure." It would be actually derogatory to our science if we were unable to obtain similar, if not better results than these empirics.

But, before proceeding to the treatment of habitual constipation and hemorrhoids, I shall first define and portray our conception of the first mentioned symptom-complex.

CONSTIPATION

We may differentiate between physiologic and pathologic constipation. Constipation is physiologic so long as the delay in the evacuation of feces produces no disturbance, not even slight, in the intestinal tract or in remote organs. These physiologic effects we have learned to recognize in other organs, for example, in the heart (physiologic bradycardia), in the female genitalia (appearance of menstruation every two or three months), etc.

Within these limits the degree of diminished activity may vary widely. Much found in earlier literature, for example, the report that patients have had only one or two evacuations in a year, may be referred to the domain of legend. But I have certainly seen a patient who had a fecal evacuation only once in four weeks, and then it had to be each time extracted digitally by a nurse.

This patient, on account of some gynecologic affection, had undergone several laparotomies; probably adhesions had formed between different portions of the intestines. In every other respect, however, her condition left nothing to be desired. Her appearance was robust, the urine was normal, and the amount of indican, in particular, was not increased.

In another case, that of a lady, aged 69, I obtained the history that the patient had been small and weak from her youth. Only once a week was there any desire to have a fecal evacuation. She had experienced no discomfort from this throughout her entire life, but she later succumbed to carcinoma of the esophagus.

Nevertheless, such diminished physiologic activity must, at least, be looked upon as a chronic anomaly and is certainly rare. Usually this results in a slow or rapid systemic disturbance, and physiologic constipation leads to true *pathologic* habitual constipation.

But we must be even more precise than this:

Not every case of chronic constipation can be designated as habitual. On the contrary, by custom this nomenclature has been applied only to those anomalies in which, in the first place, there are no other primary disturbances; and secondly, to the cases in which the intestinal evacuation as such is nowise due to mechanical or toxic effects (for example, lead colic). It is readily seen that such a limitation cannot possibly be absolute, that under the picture of simple habitual constipation all sorts of pathologic pictures, occasionally serious and prognostically unfavorable ones, may be masked. Nevertheless, we must be content, as elsewhere in the practice of medicine, with a symptomatic diagnosis so long as we have no deeper insight, so long as other decisive factors have not been added, or, finally, while the entire course of the disease receives another interpretation.

Here, at the beginning of our description, we must necessarily be suspicious of the diagnosis of habitual constipation in those cases in which the facts are not absolutely uniform and clear. In the chapter devoted to diagnosis we shall have an opportunity to justify this position *in extenso.*

ETIOLOGY

ETIOLOGY OF HABITUAL CONSTIPATION

In consonance with the above definition we can only enumerate those causes of habitual constipation in which organic changes, no matter of what nature, are absent. The number of secondary factors which induce habitual constipation is so great that an exhaustive compilation is impossible.

The causal factors of idiopathic constipation are practically important because with their removal the affection is occasionally cured.

In unbiased judgment, we must exclude a large group in which a distinct, causal etiology cannot be recognized. The affection is mild at the onset, but the intestine becomes sluggish, although the patient attaches but little importance to this until gradually the necessity for relief causes a resort, usually to purgatives, more rarely to injections. As the former gradually lose their effect, more active purgatives are employed, and the condition is thus carelessly allowed to continue year in, year out, until, finally, all remedies are inactive, or pain and catarrhal phenomena necessitate rational and comprehensive interference.

Habitual constipation may frequently be referred to *hereditary influences.* Constipation to a greater or less extent prevails in entire families. Occasionally the condition has its beginning, as I have repeatedly observed, in the first days or weeks after birth, and even in this early stage it is sometimes very difficult to combat.

This hereditary predisposition is often combined with a lack of true "*bowel training.*" The children are not taught to observe regularity in regard to their bowel movements or—and this is particularly the case with young girls—false modesty leads the children to artificial suppression. This sin is most frequently committed in schools, while upon visits, and in summer resorts.

In rare cases this form of constipation may be due to a dilatation of the colon (probably congenital), the so-called Hirschsprung's disease, the prognosis of which, even under the operation recently proposed, is very unfavorable.

A frequent cause of habitual constipation, which I have called *alimentary constipation,* is due to improper nutrition; in particular, the immoderate ingestion of meat and the avoidance of vegetables leaving a residue, as well as the use of rye bread and varieties of sugar.

The physician himself is responsible for *alimentary constipation* more frequently than is generally supposed. As an example, I may mention the diet prescribed for diabetes, which, as employed in most cases, necessarily produces constipation. A milk diet, too, as ordered in anemia, chlorosis, and nephritis, frequently has an unfavorable effect, or, at least, it does not produce the desired result because the constipation which usually follows the use of a milk diet is too little considered.

To this must be added another common form of constipation which is not sufficiently borne in mind, and, indeed, is not generally recognized: *That due to the employment of drugs.* In acute intestinal disturbances and in cholera nostras, but particularly in appendicitis, and probably also in other forms of peritonitis, in occlusion of the bowel or in pseudo-ileus, large doses of opiates or the belladonna preparations are frequently used or, more correctly, wasted, and this is followed, as I have seen in innumerable instances, by severe and most unyielding intestinal paresis. Besides opiates, substances containing tannic acid are often too long employed for diarrhea, and, occasionally even after a short time, produce constipation; in the course of years I have seen several well developed cases of this kind. We can scarcely be wrong in assuming that extremely severe changes in function are due to an abnormal tannic acid effect.

As a curious abnormality, I shall here call attention to a natural increase of habitual constipation which I have repeatedly observed in my practice: The purposely limited ingestion of food. While it does not seem plausible, many individuals, mostly neurasthenics, have an idea that by a natural limitation of the ingestion of food they may relieve their difficulties due to constipation.

A much more frequent cause, and one which requires less consideration than the foregoing, is the familiar one, *lack of exercise*, which has been much discussed, and which also plays an important rôle in the etiology of hemorrhoids, as we shall later have occasion to note. The influence of active exercise and, where this is impossible, of passive exercise cannot be doubted, yet we must be careful not to exaggerate the importance of this as a causative factor. For example, it is generally known that extreme habitual constipation is found in persons whose occupations certainly indicate no want of exercise, for example, in farmers, army officers, gymnasts, bicyclists and others. On the other hand, I have several times called attention to the fact that in certain forms of habitual constipation which will shortly be described bodily rest is much more likely to relieve the affection than forced exercise.

One of the chief causes of habitual constipation is *neurogenous*, or *psychogenous*, which is a better term. It is an almost constant accompaniment of general neurasthenia, but may occasionally present itself as a substantive intestinal neurosis.

A widely distributed type of neurogenous constipation is found in enteroptosis, the relation of which to neurasthenia cannot to-day be doubted even though their intimate connection is still a mooted question. As acute psychical factors may generate a state of anxiety, an irritation of peristalsis from nerve tracts which we refer to the splanchnic area, so by chronic irritation there may be a permanent diminution of the energy of innervation.

I recently treated Miss D., a teacher, aged 34, who, since Whitsuntide, 1901, had suffered from occasional diarrhea, swelling of the feet, pain in the left hypochondrium, insomnia and great lassitude. During her holidays there was marked improvement, but soon after resuming teaching marked nervousness and constipation simultaneously appeared. Purgatives, to which the patient, however, had never accustomed herself, were without effect. In the beginning simple enemata, and, later, even enemata of oil, were ineffectual. She ceased to teach, and the condition of the bowels steadily improved until gradually there was a profuse and spontaneous evacuation daily. Constipation reappeared only after excitement, even though of a pleasant nature.

In other cases of psychogenous constipation, the greatest variations between constipation and diarrhea may be observed.

[As an additional cause of chronic constipation, one finds in America formation of the stool is the proof of spastically contracting intestinal coils, which we shall consider later. In my experience these conditions vary greatly in the same individual. Nevertheless, continuous and extensive spastic contractions are frequently found associated with a spastically formed bowel movement.[1]

As a third form of habitual constipation that variety characterized by me as a *fragmentary bowel movement* must be differentiated. In this condition, as in the case of hemorrhoids, proctitis, or rectal carcinoma, there is marked tenesmus which compels the patient to seek the toilet every few hours, yet the abdominal pressure brings forth only a few particles of feces. I have usually observed this condition in patients suffering from intestinal neurasthenia.

Finally, we must mention a fourth form which is distinctly characterized and very frequent: *Stercoral diarrhea.* In this affection there is at first constipation which lasts from one to three days, sometimes even longer, and is accompanied or followed by severe colicky intestinal pain and rumbling, and by one or several copious diarrheic discharges; these are at first firm, then fluid, but may be diarrheic from the start. The same typical symptom-complex then reappears, varying only in severity, frequency and duration, both in the attacks and the intervals.

These are the most common and comparatively the most typical forms of habitual constipation. Besides those mentioned there are undoubtedly other modifications which cannot be described.

More necessary is the description of two important and, occasionally, very distressing and painful symptoms of constipation: *Flatulency* and *intestinal colic.* The former is either due to abnormal filling of the intestine with putrid gases which the intestinal musculature is powerless t expel, or it may be attributed to an abnormal and excessive production o gaseous products while the intestinal motility is good. In the forme case, therefore, gas production is normal but there is diminished inte tinal peristalsis; in the latter case there is an abnormal and copious g formation with sufficient intestinal motility. In the last, the bowel mov ments may be sufficient and regular.

Without doubt the first mentioned form is by far the most unpleasa and is intimately connected with the colic now to be described. But t second form, also, is no less unpleasant to the patient than to those abo him. Intestinal colic, as mentioned, may be due either to abnormal ret tion of gas (flatulent colic) or to other non-gaseous products of deco position (stercoral colic). In the former case the attack of pain af more or less time ceases with the passage of gas; in the latter, euphoria only follows a copious fecal discharge.

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[1] However, spastic intestinal contractions are by no means always patholo I have seen them also when the composition of the feces was quite normal. chronic diarrhea (not stercoral diarrhea) I also demonstrated well-developed, tracted, intestinal coils.

From this true intestinal colic we must carefully differentiate certain gastric difficulties to which Knud Faber[1] has recently called attention, and whose experiences we can absolutely confirm.

Another condition which belongs to the symptom picture of habitual constipation has, since Bouchard's celebrated researches, been designated as *autointoxication*. Headache, sometimes appearing as pressure in the cranium and sometimes as migraine, also dizziness and lassitude here play an important rôle. In fact, we cannot at once reject the theory of a causal connection here, and every physician of experience will recall many cases in which the above mentioned symptoms, particularly if not well developed, permanently disappeared on regulating the bowels.

On the other hand, it would be a manifest exaggeration to attribute to migraine all attacks resembling migraine and running their course with habitual constipation. Only too frequently it becomes my duty to dispel the illusions of my patients who suffer from migraine and refer their condition to faulty gastrointestinal digestion, and the fruitlessness of treatment directed to this condition has almost invariably shown me to be correct.

Finally, the well-marked picture of neurasthenia is frequently combined with habitual constipation, as has already been mentioned, and in these cases, objectively speaking, the neurasthenia cannot be regarded as the consequence but the cause of chronic intestinal inactivity.

Habitual constipation may also exert an influence upon the *kidneys*; thus, as Kobler[2] and Ebstein[3] have observed, and as I may confirm, occasionally slight albuminuria and cylindruria are met with. In how far we are here dealing with a mild grade of nephritis or a true coprogenous albuminuria cannot at present be determined. Kobler's investigations favor the view of coprogenous albuminuria. According to my own experience, albuminuria and cylindruria undoubtedly form the rarest complications of habitual constipation.

SYMPTOMS OF HEMORRHOIDS

Hemorrhoids, on account of their intimate connection with habitual constipation, naturally present many of the same symptoms. But the localization of the affection and the formation of varices add to the previously described picture a few peculiarities.

The explanation of these is found in the mechanical effect which they produce. As such, the atony in the lowest portion of the large intestine and in the rectum must be first mentioned. This causes stagnation of the intestinal contents in the sigmoid flexure and in the ampulla recti, and also the development of a catarrh of the rectum, occasionally even of the sigmoid flexure. Tenesmus, which is very frequent, is also due to stagnation of the intestinal contents in the rectum and in the sigmoid flexure.

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[1] *Knud Faber, Arch. f. Verdauungskrankheiten*, 1902, Bd. VIII, Heft 1.

[2] *Kobler, Wiener klin. Wochenschrift*, 1898, Nr. 20.

[3] *Ebstein*, "Die chronische Stuhlverstopfung in der Theorie und Praxis." Stuttgart, 1901, p. 86.

Occasionally the propulsion of the contents becomes difficult from a purely mechanical cause, namely, the extension of the varices and nodules. Thus a vicious circle is formed, for the varices increase the constipation and, *vice versa*, the latter condition aggravates the hemorrhoidal affection.

Another source of difficulty is the ready tendency to inflammation or even ulceration of the nodules (which have extraordinarily thin walls) from the continuous mechanical irritation to which they are exposed, this being favored by a lack of cleanliness. This gives rise to the itching and burning of which the patients so frequently complain, and occasionally also to severe, boring, unbearable pain combined with the most distressing tenesmus. Not infrequently the pain and burning sensation are felt not at the point of affection but higher up, in the lumbar region or in the shoulders, and quite typical attacks of sciatica may occur—I have seen several undoubted cases of this kind—which, if the origin of the trouble is not located, may lead to various therapeutic errors.

The most common manifestation of hemorrhoids, as is known to every layman, is the periodically appearing *hemorrhages*. These may vary in degree and frequency. It was formerly assumed that the hemorrhage from hemorrhoids was beneficial, and to some extent a self-regulation of the condition of stasis in the portal vein circulation.

Upon unprejudiced testing we must admit that there is at least a grain of truth in this view, inasmuch as, after diffuse depletion, there is a transitory decrease in the varices distended with blood and, hand in hand with this, euphoria of shorter or longer duration may appear. Beyond this there is no salutary influence from the bleeding of hemorrhoids; for, in the first place, the expected amelioration from loss of blood does not always follow, and, moreover, from their duration and intensity, the latter may produce severe anemia without in the slightest degree diminishing the local affection. The proverb, "All that glitters is not gold," is also true of "the golden vein." The formation of hemorrhoids is intimately connected with enlargement of the liver. I cannot express this relation in figures, but all who are accustomed carefully to percuss and palpate the liver of patients with hemorrhoids must have noted the frequency of hepatic congestion.

One of the most positive and interesting cases of this combination is the following: A hotel proprietor, aged 40, but an exceptional example of temperature in regard to alcohol, for many years had been suffering from habitual constipation and highly developed external and internal hemorrhoids. The liver was four fingerbreadths below the margin of the ribs; there was no enlargement of the spleen. The appetite was good, the patient digested all food without difficulty. From time to time the enlargement of the liver increased decidedly, and caused a feeling of pressure and tension in the abdomen. Whenever hemorrhage from the hemorrhoids appeared, the liver decreased in size, and the symptoms disappeared. Instead of bleeding from the hemorrhoids sometimes there was very copious hematemesis; no symptoms of ulcer or gastritis were ever observed, but in the course of time there were six severe attacks of hematemesis. Without forcing scepticism too far, we are here actually dealing with a case of *vicarious gastric hemorrhage*. This, therefore, is not, as v. Schrötter[1] has recently maintained, a canard (Räubergeschichte).

Earlier literature, which found fanatic expression chiefly in the teachings of Georg Ernst Stahl, ascribed to hemorrhoids a far greater importance than that just alluded to. According to this, the stoppage of hemorrhoidal bleeding was said to produce all sorts of internal diseases (gout, diseases of the liver, the heart, the lungs, and others). There can be no doubt that this teaching over-reached its mark, and was justified only in so far as congestion in the deeper areas, occasionally also in organs situated higher up, was affected by the portal vein circulation.

DIAGNOSIS

DIAGNOSIS OF HABITUAL CONSTIPATION

In turning to the diagnosis of habitual constipation and hemorrhoids, the recognition of these affections at first sight appears very simple, and to require no special elucidation. In fact, this is true in the overwhelming majority of cases. But even this apparently simple clinical picture may cause perplexity in diagnosis, and it is therefore absolutely necessary that patients complaining of *habitual constipation* be examined just as thoroughly as those suffering from any other affection.

The important question to be decided in every case is this: Is the constipation habitual, viz., primary, like that previously described, or is another causal factor present? At the beginning of this article we called attention to the numerous causes which may produce this condition; possibly their rôle is not always important, but a thorough diagnostic insight is also a valuable aid in the treatment.

At this point we shall busy ourselves especially with the various diagnostic difficulties which are referable to the digestive apparatus itself.

It is a common experience to find that patients with various disturbances of digestion regard habitual constipation as the most prominent and frequent cause of these, which occasionally, however, masks other important symptoms. Thus, I have frequently noted that patients with stenosis of the pylorus, even with gastric carcinoma, complain merely of habitual constipation, while the serious underlying gastric affection is revealed only by a careful examination.

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[1] *v. Schrötter, Verhandl. des Congresses für innere Medicin*, 1902. "Discussion zu dem Vortrage von Ewald, Ueber die Diagnose des Magengeschwürs."

The possibility of deeply situated intestinal disease is more significant. First in importance is carcinoma of the intestine, especially that involving the colon, not on account of its frequency, but because it may lead to grave errors. Of the entire structure of the symptom-complex only constipation can be discerned upon careful, and especially upon superficial, examination. What is true of carcinoma is also true of benign stenosis. A less serious error, but nevertheless not without interest, consists in the non-recognition of the combination of habitual constipation with colitis, to which we shall refer later.

These considerations, which by no means exhaust the differential diagnosis, sufficiently indicate that the diagnosis of habitual constipation must, under all circumstances, be preceded by a thorough local and general examination.

The special diagnosis which then follows must comprehend the physical investigation of the intestines and the feces.

In the former, the recognition of intestinal atony by filling the bowel with water or gas is easy. The same condition is pointed out by the finding of contracted intestinal coils, especially of the transverse colon, the cecum and the sigmoid flexure, which also indicate the spastic character of the chronic constipation. Extreme meteorism favors stagnation of the contents; on the other hand, sensitiveness to pressure in the region of the colon is not common in simple habitual constipation, but indicates colitis.

The examination of the rectum is an integral part of an examination of the intestinal canal. Aside from the discovery of hemorrhoids, which will be fully described later, angular bulgings will probably be observed which indicate stagnation of the feces in the lower portion of the bowel. If large scybala are recognized in the rectum, the correct remedy may be at once employed.

In conclusion, observation of the bowel movement itself is a valuable aid in the recognition of the nature and the extent of the constipation. Particles of mucus should be examined, provided a so-called *exploratory irrigation of the intestine* is not undertaken; this I always advise, particularly if the patient reports the appearance of mucus, or if there is sensitiveness to pressure over the colon.

For the physician not familiar with intestinal diseases it may here be remarked that irrigation of the intestine is carried out in a manner similar to gastric lavage. For this purpose a soft rectal tube is attached by means of a small glass tube to a rubber tube about a yard and a hal long which has inserted, at its upper end, a glass funnel. The funnel is elevated and water at a temperature of about 30° C. is permitted to flow into the intestine; upon lowering the funnel the fluid is returned and received in a vessel held for this purpose. This fluid may then be readily examined for mucus and other pathologic constituents.

DIAGNOSIS OF HEMORRHOIDS

The *diagnosis of hemorrhoids* necessitates a local inspection of the anus, and a *digital examination* of the rectum. This examination, which is really self-evident, is unfortunately, notwithstanding the urgent advice given in text-books and medical journals, not sufficiently often made. The operative treatment of rectal carcinoma, which is too often unrecognized and sails under the flag of hemorrhoids, would then give us much more favorable results.

In every case of hemorrhoids it must also be determined whether we are dealing with *external, internal,* or *mixed* hemorrhoids. While the first are readily recognizable, the diagnosis of internal hemorrhoids may occasion great perplexity. Internal hemorrhoids are most readily revealed by strong pressure, and best after injections of a hot saline solution or glycerin.

Where nodules do not thus appear, and the bleeding from the anus indicates a hemorrhoidal origin, it is possible, as J. Schreiber[1] has lately shown, to determine the source of the hemorrhage by the rectoscope, and to control and treat it surgically. Similar cases have been described by Nothnagel[2] and Ewald.[3] According to Schreiber, hemorrhoidal venous dilatation is observed much higher in the intestine than was previously thought possible, even as high as the sigmoid flexure.

The differential diagnosis and the complications of hemorrhoids require a few brief remarks. In regard to the former, it must be emphasized that the confusion of hemorrhoids with other diseases of the rectum, provided the rectum is carefully palpated, is very rare. At most small polypi or angiomata may cause perplexity. The differentiation of a beginning carcinoma from ulcerating hemorrhoids is sometimes difficult, all the more so since from these cancroids may develop in the course of time. Successive palpations and rectoscopic examinations, perhaps the excision of a portion under narcosis or local anesthesia, will in most cases clear the situation.

The *complications* of hemorrhoids are numerous. Catarrh of the rectum (proctitis haemorrhoidalis) has already been mentioned; this frequently runs its course with tenesmus. On strong pressure we observ as a rule, the discharge of a gelatinous yellowish-white mucus.

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[1] *J. Schreiber,* "Die Rectoromanoskopie." Berlin, 1903, p. 73 et seq.

[2] *Nothnagel,* "Zur Klinik der Darmkrankheiten." *Festschrift z. Feier d. 60. Geburtstages von Max Jaffé.* Braunschweig, 1901.

[3] *Ewald,* "Klinik der Verdauungskrankheiten," 1902, III, pp. 425 u. 426.

In the course of hemorrhoids more or less deep ulceration ofte occurs and causes severe burning or pain. Careful examination with th speculum will usually enable us to recognize this complication.

Occasionally there is incontinence of the sphincters of the rectum, which even without the prolapse of nodules, leads to [illegible] disturbances, par ticularly to the discharge of thin, fluid feces. Inspection and digita examination will readily reveal this condition.

When hemorrhoids have persisted for a long time, hemorrhoidal pro lapse frequently develops and may vary greatly in severity. In the milde cases prolapse only occurs upon defecation. In more advanced cases i may follow any movement, for instance, sneezing, the passage of flatus, et

TREATMENT

TREATMENT OF HABITUAL CONSTIPATION

Where [illegible] long treatment, vari ous methods [illegible] habitual constipation are [illegible] physicians. [illegible] different measures [illegible] various curative agen [illegible] and [illegible] effectiveness of [illegible] as they ar [illegible] cases. [illegible] therefore [illegible] degree of consti pation [illegible]

[illegible] to be the first [illegible] means [illegible] many patient [illegible] measures [illegible] that they [illegible] sometimes [illegible] time, that is, for [illegible] measures are [illegible]

[illegible] the degree [illegible] remedies [illegible] difference [illegible] these are [illegible] and among the [illegible] compound licorice powder, flowers of sulphur and the like—in small doses are sufficient, sometimes for years; these cases are, therefore, unquestionably mild. In others these remedies are without result or are effective only in very large doses. Such cases may be designated as moderately severe. The most extreme degree of chronic constipation is represented by the patients in whom the neutral salts, and even drastics in large doses, either fail to bring relief or act insufficiently. But another group is also to be differentiated, and in these neither mild nor drastic purgatives produce free action, while the simple injection of water at once gives relief. These, apparently, are the cases in which the lower portion of the intestine is the seat of intestinal atony, and here we must bear in mind the psychogenous or neurogenous group in which purgatives, injections, and massage, etc., are without effect unless supplemented by the complete removal of the patient from his former surroundings and the cares of his occupation.

Only by such a separation can we decide as to the nature and degree of the affection, and only thus can we correctly estimate the value of the many remedies and methods employed to relieve the condition.

Before proceeding with our discussion it is by no means superfluous for us to ask: What is meant by the cure of habitual constipation? In my opinion we can only speak of habitual constipation as cured if, a year after the termination of the treatment, there is a spontaneous and ample evacuation every twenty-four to forty-eight hours.

If all physicians were to accept this as a standard, our confidence in the efficacy of measures which have apparently been very successful would soon be shattered.

As soon as we lower our standard, and regard temporary improvements as cures, the number of successful remedies is, of course, very considerable.

In the following, the treatment of habitual constipation will be considered only from that standpoint which we have established as indicating a cure.

As everywhere in medicine, *prophylaxis* plays the leading and most important rôle. It begins in childhood with the consideration of the previously mentioned congenital predisposition to constipation. In addition to this, it must be the object of home training and of our professional endeavors to instruct children as to the importance of regulating their intestinal functions. To attain this end a thorough understanding of the process of nutrition is, above all, necessary.

We know that, especially in the better classes, the desire for a strengthening diet takes precedence of all other considerations, and the mistaken parents regard expensive meat and the nutritious egg as the only nourishing, or, at least, the best food.

Aside from the fact that a too stereotyped diet fails to produce the nutritive effect expected (to the astonishment of those who institute it), another undesirable factor is added—habitual constipation. It is therefore the duty of the physician to regulate the nutrition of children, and from time to time to add to the uniform, constipating diet food which favors the regular action of the bowels.

What is true of children also applies to the condition in adults designated by me as alimentary constipation. In particular, the table d'hôte with its abundance of meat and fish in contrast with its usually small amount of vegetables is a frequent cause of constipation in young persons.

Here, in a restricted sense, the same laws of treatment are operative which, in Germany at least, Albin Hoffmann first utilized with such marked success in therapy: *exercise* and *abstinence*. In alimentary constipation exercise is the more necessary; upon this, in fact, are based our most important methods of treatment, which range from ordinary walks to Swedish movements and massage. A practical and even a decided influence is to be ascribed to the diet inasmuch as here thermic, chemical and mechanical stimulation, occasionally all combined, stimulate peristalsis to an increased activity. *As a practical, pioneer factor, the diet, therefore, occupies a leading place among the curative agents.*

In the first place, the diet of chronic constipation, like the diet in other affections, is based upon the law of experience. To the present time the sanction of inductive science is lacking. Nevertheless, we may picture to ourselves more or less completely the effect upon the intestinal function of an increase in diet. I designate this as *constipative diet*.

It is generally known that certain foods stimulate and others inhibit intestinal activity just as do certain drugs, and upon exact investigation the line of demarcation between the effect of a drug and that of the food is almost obliterated. Whether I administer tannic acid in the form of acidum tannicum or as acorn coffee, whether huckleberry wine or red wine containing a large amount of tannin, whether rhubarb compote or an infusion of the root of rhubarb, whether I give fig syrup or half a dozen fresh figs certainly makes no fundamental difference; at most, in the one case we are employing the raw substance, and in the other we are administering the active principle in as pure a form as possible.

The physiology of food gives us a large number of such remedies which stimulate the intestine. We may divide them into the following groups:

1. Substances containing sugar.—To this group belong common cane sugar, milk sugar, honey, preserves, [illegible] whipped cream fruits containing large [illegible] sugar, such as [illegible] figs, oranges dates, etc. The effect of sugar is [illegible] In the first place solutions of sugar in the stomach produce [illegible] dilution secre-

tion, Strauss), and in the sugar not at once absorbed acids of fermentation develop.

2. *Foods containing organic acids.*—Among these are: Buttermilk, sour milk, kefyr, kumiss, sour whey; also fruit wines, for example, cider or Moselle wine, currant wine, gooseberry wine and, in Russia, the so-called "kwass"; sour lemonade (made from lemons), acid fruits (apples, sour cherries, gooseberries and currants, etc.); rye bread, characterized by its amount of sour dough, belongs to this category.

3. *Salty substances.*—As the commonest remedy, ordinary table salt in its various combinations (salt water, salt herring, caviar, sardellen, etc.). Smoked and pickled foods also belong to this category.

4. *Substances containing carbonic acid or those which form carbonic acid.*—In this group belong the natural or artificial waters which contain carbonic acid, also the simple alkaline waters, and the above mentioned kefyr, kumiss, kwass, matzoon, etc. Yeast and yeast bread may also be included in this category. Finally a number of so-called gas-forming vegetables; these produce CO_2 as well as other varieties of gas.

5. *Substances which contain fat, and in which the amount of fatty acid assists the action.*—The most common remedies here are butter, olive oil, sesame oil, or linseed oil, which is generally and quite properly, I think, regarded as difficult of digestion. Perhaps the value of linseed (see later) depends upon the large amount of fat it contains. Here, on account of its contents of fat, the so-called mayonnaise is to be included, as well as Italian salad and, perhaps, all salads prepared with oil.

These are the main chemical stimulants to the intestine, and to these must be added the thermic and mechanical ones.

6. On account of its thermic effect only *cold,* naturally, must be considered. This is best employed as a drink in the form of cold water, cold milk, either sweet or sour, cold lemonade. Cold soups (such as fruit soups, and the like) have, in addition to other effects, also a certain thermic influence. On the other hand, cold or frozen fruits, because usually introduced into a full stomach where equalization of temperature soon occurs, are less beneficial. The action of these substances depends mainly upon the state of the stomach, whether full or empty, and the optimum of the thermic effect is consequently found in an empty stomach, a fact daily demonstrated in practice.

7. The *mechanical* effects of food are manifold. The more the indigestible residue from a food, *cæteris paribus,* the more it promotes the passage of the intestinal contents.

The greater the proportion of food absorbed in the intestinal canal, the less suitable is it in the treatment of habitual constipation.

We are indebted to the researches of Rubner for our knowledge of the absorption of food; if not of all at least of the most common ones. According to these the percentage of non-absorption is as follows:

	Of the dried substance	Of albumin	Of carbohydrates
In broiled and fried meat	4.9–5.3	2.0–3.6	...
In shell fish	4.8	2.5	...
Hard boiled eggs	5.2	2.6	...
Milk	8.8	7.8	...
Wheat bread from fine flour	4.2	21.8	1.1
Rolls	5.6	22.2	2.9
Rye bread from coarse ground corn	13.1	36.7	2.9
Rye bread from the whole corn	20.9	46.6	14.3
Pumpernickel	19.3	43.0	13.8
Rice	4.1	20.4	0.9
Peas	9.1	17.5	3.6
Green beans	15.0	....	...
Beans	18.3	30.2	...
Mashed potatoes	9.4	30.5	7.4
Savoy	14.4	18.5	15.4
Yellow turnips	20.7	39.0	18.2

This Table is very instructive for our purpose. It shows the almost complete absorption of animal substances. The digestibility of milk in adults is only slightly less than in infants, which would scarcely be supposed from its composition, but there are probably individual differences.

The absorbability of vegetable substances is far less than that of animal substances. As is shown by the Table, much of this depends upon the mode of preparation. In materials which are baked the addition of coarse bran lessens absorption; finely ground, however, it increases absorption. Cellulose in the potato is readily digested, and best the cellulose of young leaf vegetables. In all of these carbohydrates the absorbability is largely dependent upon the manner of preparation.

As is also evident from the Table, bread made of rye, ground from the whole grain and baked with yeast, and pumpernickel show the lowest degree of absorbability. Among vegetables, the same is true of beans, Savoy cabbage, yellow turnips. It is to be regretted that the degree of absorbability of other vegetables and other varieties of cabbage is not given in Rubner's Tables. The latter, however, may generally be reckoned among the carbohydrates that are only slightly absorbed.

Among substances which exert a mechanical action belong all products with skins, membranes which are usually eaten raw, but sometimes also cooked, and which are very imperfectly digested. Among these are all the different fruits, in so far as they are unpeeled, among the legumes peas and beans, and among other vegetables asparagus in particular. Mushrooms are not at all digested; probably to the same category belongs the radish. Complete absence of digestion also characterizes mustard seed and linseed. These, therefore, are occasionally employed with success as coarse, mechanical, euperistaltic remedies. However, the possibility of the entrance of mustard seed or linseed into the appendix may be regarded as a source of danger.

We note, therefore, from this description that, in a broad sense, the diet contains all the potentials which are necessary to affect a sluggish intestine, and that these are occasionally combined with one another in a most remarkable manner. For example, in fruit we find sugar, acid, cellulose and pectin. The methods of serving these also produce combinations which assist intestinal activity; for example, sour milk with sugar and rye bread, cold cereals, sugar, currants and hyperfermented beer.

We are thus enabled to find a number of laxative remedies from which we must select the best and most effective, and combine them.

It must be primarily emphasized that in cases of advanced habitual constipation that are at all stubborn, any single remedy to stimulate intestinal activity will usually be without effect. If, however, we combine several of these, for example, by administering sugar, acids, table salt, oil, material rich in residue, in proportions adjusted to the mode of life and the habits of the patient, *the total effect equals that of a powerful purgative.*

It is obvious that in the choice of these natural purgatives we must individualize very strictly. In addition to certain idiosyncrasies or bad habits which must be reckoned with in practice, the state of the gastrointestinal canal comes into question. Acid and fatty substances are frequently not well borne; they may produce pyrosis and a sense of gastric oppression. In other cases coarse bread and pumpernickel generate gas, and other products must occasionally substitute for the varieties of sugar and fruit. Certain disturbances in metabolism, for example, gout, obesity and diabetes, may necessitate a reduction in amount.

This apparent or actual intolerance should by no means force us to abandon the treatment, for the dietetic laxatives which remain may be given in increased quantity. On the other hand, inversely, the diet must be so regulated that secondary disturbances, for instance, anemia, chlorosis, under-nutrition, and enteroptosis may be favorably influenced by it. In such cases the proteids, sugar, and fat should be particularly prominent in the diet.

A significant point in the treatment of habitual constipation is the strict avoidance of astringent substances which inhibit peristalsis. Self-evident as this appears to be, in my experience such errors are frequently committed. Thus, for example, a patient with constipation is placed upon a very suitable diet, but, at the same time, is permitted to take cocoa, red wine, rice or fine wheat bread, or, what is equivalent, these foods are not strictly prohibited. Attention need scarcely be called to the fact that a diet for constipation is thus made absolutely of no effect.

Concerning this somewhat complicated system the question may be and has been asked: Why institute such a tedious treatment when we may more quickly attain the same results with other curative remedies?

Were this actually the case, the dietetic treatment of habitual constipation would, in truth, be an unnecessary hardship. Later on we shall discuss other valuable remedies which increase intestinal activity. But, in considering the results of dietetic treatment, and out of a rich experience, I deduce the following:

The effect of the constipation diet is, in the overwhelming majority of cases, very rapid, and appears, as a rule, even within the first few days. In most cases this result is permanent, and the patient gradually resumes his normal diet. We then consider this an *actual cure.* In the minority, however, after weeks or months the effect apparently ceases; the patients respond imperfectly, or not at all, to the diet. We see, therefore, the same necessity of adapting the stimulation to the case as in a treatment by purgatives. Occasionally, however, this diminution of stimulation is not due to the cumulative effect of the diet, but to the fact that in the course of time the patients fail to adhere to it. Occasionally the treatment is interrupted by prolonged traveling, by improper food in hotels, by psychical emotion or other unfavorable circumstances, and it then becomes exceedingly difficult to stimulate afresh the impaired intestinal function. In conclusion, there are patients in whom a regulated diet acts only so long as they adhere rigidly to all of its details; even slight modifications produce disturbances. In these cases a cure such as I mean is out of the question.

These exceptions, however, cannot alter the fact that the effect is permanent in a large number of cases. *To say nothing of purgatives, such a permanent cure never takes place otherwise nor by any other remedy.*

For this reason dietetic treatment of the mild as well as of the severe forms of habitual constipation must form the normal process. When this is not effective, other remedies are to supplement it, but by no means to substitute for it. Following this is a *diet list* for an ordinary case of habitual constipation, the condition of the stomach being considered normal:

In the morning upon an empty stomach: A glass of salt water or sugar water.

8 o'clock: Coffee with milk, one tablespoonful of milk sugar (or manna or levulose), 50 grams of Graham bread, or D.K. bread, or Simon's bread 10 grams of butter, 20 grams of honey or sweet marmalade.

11 o'clock: 250 grams of sour milk, or buttermilk, or kefyr 2 day old; 50 grams of Graham bread, or D.K. bread, or Simon's bread; 1 grams of butter (1 egg[1]).

2 o'clock: 50 grams of potatoes (mashed potato or with the skin 200 grams of vegetables,[2] 150 grams of meat or fish, 50 grams of sal

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[1] With a good constitution this may be omitted.

[2] With a weak stomach as purée; with a good stomach, in the natural form.

(with lemon and oil); about 100 grams of stewed fruit (sweet or sour), 150 to 200 grams of cider, Moselle wine, or lemonade; raw fruit.

4 o'clock: Coffee with milk or whipped cream, 50 grams of marmalade, 25 grams of Graham bread or Graham biscuit.

8 o'clock: 100 to 150 grams of meat or fish (cold or warm); salad (see above); 50 grams of stewed fruit; soft cheese; 50 grams of Graham or rye bread, or pumpernickel with butter; 100 to 150 grams of cider or Moselle wine.

Before going to bed: Raw fruit of various kinds (according to season) and in desirable quantity.

We add here, as an example of a variation in the diet, the following which may be given in a mild case of *diabetes*:

Upon an empty stomach: A glass of salt water.

8 o'clock: Coffee (black); 30 grams of manna, 20 grams of Graham bread, 30 grams of butter.

11 o'clock: 250 grams of sour milk (buttermilk, kefyr); eggs (1 to 3), also scrambled eggs with considerable butter or bacon.

2 o'clock: About 200 grams of vegetables (spinach, string beans, cauliflower, Brussels sprouts, asparagus, artichokes, sweet potatoes); about 50 grams of salad (with lemon and oil), about 100 grams of diabetic stewed fruit; 200 grams of cider or Moselle wine; radishes; butter and cheese according to desire, 25 grams of Graham bread, and two apples.

4 o'clock: Coffee without milk, 50 grams of manna, 50 grams of *bitter* marmalade.

8 o'clock: 100 to 200 grams of meat or fish (even fat varieties, warm or cold); salad (as above) or mayonnaise; diabetic stewed fruit; 50 grams of pumpernickel; 30 to 50 grams of butter; cheese (as desired); Moselle wine; radishes.

Before going to bed: Two apples.

In this diet the amount of carbohydrates is not very considerable, but it may be even further reduced. Manna, which acts as a stimulant even in the severest forms of diabetes, undergoes complete combustion. Attention is called to the large amount of fat in the food which serves the purpose of stimulating intestinal activity.

If there be a predisposition to obesity, the fat should be omitted from the diet, and this, as a rule, will produce a sufficient reduction.

In the given case other curative methods may be added to these dietetic rules, and this will ensure a gratifying result. Although there can certainly be no objection to this, yet, from a critical standpoint, it is advisable first to test the effect of one method, and to employ a second only when the first has failed.

With this constipation diet marked flatulency is frequently observed, even when its effect is otherwise very good. By a suitable change in the details this may be avoided without decidedly lessening the effect. Occasionally, however, as I have observed, a certain self-adaptation of the intestine is noted. The fermentation, previously marked, gradually ceases, and occasionally this cessation appears to be sudden.

As further aids, we most frequently resort to the following: *Abdominal massage, gymnastic exercises,* and *electricity.*[1]

In regard to *the first* of these we must discriminate accurately, as in the results of the diet, between temporary success and permanent cure. That the former is often attained, even with surprising rapidity, there cannot be the slightest doubt. But, in my experience, a permanent cure, even in cases not too far advanced, i. e., those which promptly respond to dietetic treatment, does not so frequently occur. At all events, the cases of cure of severe habitual constipation exclusively by massage are certainly rare. It might be urged in opposition to this that massage was unsuitable in these cases, or, perhaps, that it was not long enough continued. This is, however, not the case. I have seen striking failure after very thorough and long-continued massage.

The results of *electric* (*usually faradic*) treatment are, in my exper ence, more favorable. For this purpose we employ only faradization the abdominal walls by means of broad, flat electrodes, or, even bette by means of massage rolls, or, what is perhaps best, intrarectal faradiz tion by means of soft rectal sounds composed of Nelaton rubber.[2] T treatment, at first, should be for about ten minutes daily, and with i creasing improvement may be only every second or third day. In t course of time the patients frequently learn to apply the current the selves, or with the aid of some one at home; a factor which is not to undervalued in comparison with massage.

Whether severe cases of habitual constipation can be cured either faradization of the abdominal walls or by intrarectal application appe to me to be doubtful. But I can positively assert that the combinat of faradization with the diet elaborated above will generally bring ab a permanent recovery. Unquestionably relapses will occur and—altho rarely—failures. In my experience a point of practical importance is t if no results are obtained by rectal faradization within the first two we they do not follow even with continued treatment.

As massage and electrotherapy have found numerous adherents, so have *gymnastic exercises,* indoor gymnastic exercises (Schreber's cur gymnastics) as well as mechanical gymnastics (Swedish curative nastics). In how far these methods are permanently effective wit the aid of others I cannot state, as I have no personal experience. they assist in a greater or less degree cannot be doubted.

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[1] It is not our object to enter here upon the technic of these methods, a limit ourselves to a critical analysis of their curative value.

[2] *Boas,* "Diagnostik und Therapie der Darmkrankheiten." Leipzig, 1901, 2. page 178.

Among the methods of treatment most frequently employed, and also misemployed, is *hydrotherapy* in its various modes of application. In the atonic forms of habitual constipation, cold Sitz baths, or half baths (22° to 20° R.) with high affusions over the abdomen, as well as rain baths with a movable stream upon the abdomen, are of use. In the spastic form protracted full baths of warm water (30° to 32° R.) or warm Sitz baths are advised. The warm douche, the Scotch douche, warm compresses to the abdomen, application of hot tubes, steam compresses, and thermophores are all beneficial. In obstinate atonic constipation I have occasionally seen remarkably good results from the application of an ice-bag to the abdomen.

In one case, which was extraordinarily tenacious, there was a profuse evacuation, contrary to all expectation, when the patient (who was suffering from purulent cholecystitis) had an ice-bag applied over the hepatic region. After the cessation of the attack constipation reappeared. On the repetition of the attack of cholecystitis, an ice-bag was again applied, and normal bowel movements again took place.

Such hydropathic treatment is generally indicated in cases of intestinal neurasthenia; but, on careful observation, we must admit that failures under this form of treatment are by no means so rare as some enthusiastic hydrotherapeutists report.

Up to a certain degree, some varieties of sport are included among the physical methods: Bicycling, rowing, tennis, golf and others. They may also assist the curative action of other remedies.

We now come to the important, and most popular, form of treatment: *treatment by purgatives.*

The value of purgatives—of this there is no doubt—is, according to the nature of the case, chiefly symptomatic. Nevertheless, we note in some cases that mild or strong purgatives are taken for years, and even decades, without injury to the patient and—what is more important—they do not lose their effect. As a rule, however, such a permanent effect is not often seen.

Usually the condition is such that patients rapidly exhaust the entire range of purgatives, from the mildest to the most drastic; and it not rarely happens that even these are ineffectual unless taken in dangerously large doses, when, accompanied by severe pain, they produce the desired result.

These experiences which are constantly repeated in tiresome monotony give to the true purgatives a merely secondary position in the treatment of habitual constipation. I employ them only under two circumstances: In cases of constipation so obstinate that dietetic or physical remedies have no, or but very slight, result. But in such cases I also utilize the powerful effect of the constipation diet, and supplement it by the administration of a mild laxative in the smallest doses, or by suitable injections. I use [illegible] commonly in the [illegible] of the aged, i. e., [illegible] At the age we may expect to use [illegible] difficulty. I [illegible] purgatives to [illegible] employed in stubborn cases, such as those of Kissingen, Friedrichshall, Hunyádi-Janos, Saidschütz, Montmirail, Apenta, etc.

It is evident that mineral water cures are nothing more than purgative cures, and that from these alone permanent relief (in our sense) is not to be expected. Therefore, cases of habitual constipation should not be sent to such spas with the idea that the treatment there forms the main indication. In such cases, the patients return from the fatiguing and expensive journey much disappointed; a condition which I see every season. On the other hand, mineral water cures, like laxatives, may heighten the effect of proper dietetic treatment or may lessen and even remove some of the complications of habitual constipation, for example, cholelithiasis, hemorrhoids, and the symptoms of so-called autointoxication.

Unfortunately, in the realization of this hope the diagrammatic effect of the spa treatment with its monotonous, bland diet is rather inhibitive than effective. In this respect, the spa physician should try to modify the traditional and uniform dietary in force at the different resorts.

Injections form a valuable auxiliary in the treatment of habitual constipation. It is well to differentiate between occasional enemata and *retained* enemata; both have their distinct indications and proper places. The first serve to dislodge by a single, quick stimulation masses of feces impacted in the lower portion of the bowel. For this purpose we may combine chemical or thermic stimulants with the fluid used for injection.

Since, however, in these cases, as the name indicates, the action is to be rapid, medicaments are of little importance. We may rather look for in effect from thermic agents. For this the rule is to employ occasional njections, preferably without additions, about one-half to one liter of cool r cold water. The smaller the quantity used the better the result, as, therwise, an immoderate distention of the already paretic musculature ay easily be produced.

Much more suitable, and probably more efficacious, are *retained* emata. They are indicated in atony of the entire colon, and, inversely, mical additions are very suitable, particularly such as have a tendency dually to soften the fecal masses, while the low temperature may read- cause an abnormally rapid peristalsis, and, consequently, only partial uation.

Among valuable retained enemata we must first mention *those of oil* h have been much employed by Fleiner's advice. The action of these oil ata is not, as might be supposed, coprolytic but antispasmodic. They the abnormally excited muscular action of the intestines, and thus ce normal peristalsis. The chief indication for oil enemata is, there- in spastic constipation, although satisfactory results are now and ttained also in the atonic form.

oil enema should consist of not more than 200 to 300 grams, and ould remain in the large intestine about ten hours. Larger quanpurgatives extensively in the *constipation of the aged,* i. e., in persons beyond sixty years of age. At this age we may expect to use moderately strong purgatives for years without experiencing difficulty. In the last, or prior to the last, decade of life I also prefer purgatives to injections as being more simple. It is presupposed in this that the patients have not as yet exhausted the whole armamentarium of purgatives.

In regard to the choice of purgatives in the individual case, mild purgatives have for a long time been distinctly separated from true drastic agents. The latter are never employed in the treatment of habitual constipation unless complications occur in the course of the disease. A great number of mild remedies are at our disposal, so great that the physician, no matter how numerous his patients, will scarcely ever be in a position to utilize all the drugs.

In my experience the following remedies are quite sufficient: Rhubarb in its various forms, magnesia, effervescent magnesium citrate, Carlsbad salt, flowers of sulphur, compound licorice powder, extract of cascara sagrada (in the form of the fluid extract or in pill), the preparations of tamarind and, finally, among the newer remedies, purgen (0.5 three to four times daily). Preparations of *aloes, podophyllin, colocynth, bitter waters and bitter salts, castor oil, and many others should be relegated to oblivion in the treatment of habitual constipation: They do more harm than most physicians believe.*

The different laxative teas commonly used by the laity (Gastein tea, Hamburg tea, Hartz Mountain tea) and similar compositions are merely arbitrary and wholly superfluous combinations of various active laxatives, the effect of which must, naturally, vary according to the dose and preparation.

More suitable and simple laxative teas for domestic use are, for example, alder or senna tea (and also cassia); the herbs are placed in cold water and then allowed to boil. I occasionally employ the latter in this form, and it acts very profusely and without causing griping.

In employing either of these remedies it is well, as Einhorn advises, gradually to decrease the dose; this may be done in many, but not all cases. In any event, it should be our object not to use laxatives unnecessarily, and it is wise to continue to use *the same remedy,* even though in increased doses, as long as possible.

Combined with a laxative therapy is the employment of *mineral waters.* Among these sodium chlorid and Glauber salt, occasionally also Epsom salt, play the main rôle. Possibly the action of these spring waters is more or less increased by the amount of carbonic acid which they contain. As representatives of the first group we have the so-called *sodium chlorid waters* (Kissingen, Homburg, Wiesbaden, Mergentheim, Soden). *Glauber salt* is represented by the hot springs of Carlsbad, the cold springs of Marienbad, Tarasp, Elster, and Franzensbad. *Epsom salt springs* may be employed in stubborn cases, such as those of Kissingen, Friedrichshall, Hunyádi-Janos, Saidschütz, Montmirail, Apenta, etc.

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Much more suitable, and probably more efficacious, are *retained* enemata. They are indicated in atony of the entire colon, and, inversely, chemical additions are very suitable, particularly such as have a tendency gradually to soften the fecal masses, while the low temperature may readily cause an abnormally rapid peristalsis, and, consequently, only partial evacuation.

Among valuable retained enemata we must first mention *those of oil* which have been much employed by Fleiner's advice. The action of these oil enemata is not, as might be supposed, coprolytic but antispasmodic. They quiet the abnormally excited muscular action of the intestines, and thus produce normal peristalsis. The chief indication for oil enemata is, therefore, in spastic constipation, although satisfactory results are now and then attained also in the atonic form.

The oil enema should consist of not more than 200 to 300 grams, and this should remain in the large intestine about ten hours. Larger quantities are superfluous, and are expelled more rapidly than is desired. Inversely, we may also obtain results with smaller quantities, and it is therefore advisable gradually to decrease the quantity of oil. Occasionally, from the beginning, smaller quantities of oil (50 to 100 grams) will suffice.

Fleiner attaches great value to a neutral oil, and for this reason advises the purest olive oil. In my extensive experience other less refined oils are just as useful, for example, poppy-seed oil, palm oil, linseed oil, rape oil, and others. Sesame oil is less costly than olive oil, and just as free from fatty acid, while peanut oil, lately introduced into commerce, is good and cheap.

One disadvantage of the treatment by oil enemata is the soiling of the body and bed linen, which cannot be prevented even by the greatest care. An attempt has therefore been made to substitute other active agents for oil enemata. Among these I have advised emulsions of soda, castor oil and cod-liver oil. Soap water may also be emulsified with oil and glycerin, and various additions may be made to these emulsions (castor oil, gum arabic, honey, etc.).

In my opinion it is best to give the retained enema late at night when absolutely at rest, i. e., while the patient is in bed. The injection should be given by means of a simple irrigator and a soft rectal tube. When experience has shown that the oil is not retained, the irrigation tube may be placed in a glass or porcelain vessel so that the escaping oil may flow out without soiling the bed-linen. I have rarely made use of this method.

Instead of an irrigator, large glass syringes, or even more complicated apparatus may be used, but in none of these instruments do I see any particular advantage.

Although the treatment of intestinal peristalsis by oil and emulsion enemata gives excellent results, yet this *by no means effects a cure of habitual constipation.* The method is only palliative. If the treatment is suspended after a short time, intestinal activity also ceases, but unquestionably, as a palliative method, the treatment by oil, although not convenient, is certainly the most effective method. It has the additional advantage that it does not lose its effect by long-continued use, and in this respect is superior to internal laxatives.

Instead of large injections, so-called minimal enemata may be used, Formerly Oidtmann's purgative, which consisted chiefly of glycerin, was considered very useful. In fact, glycerin in small doses (2 to 5 grams) will liquefy fecal masses in the lower portion of the intestine. In 1887, instead of glycerin enemata, I employed glycerin suppositories, i. e., hollow cones made of cacao butter and filled with glycerin, which since then have been much used, and are, unfortunately, often found in commerce in improper form.

Glycerin and glycerin suppositories may have an irritating effect; in hemorrhoids they sometimes increase the tendency to bleeding, and, under these circumstances, are contraindicated. When there is fecal accumulation in the rectum, however, they are valuable.

Hiller[1] and, lately, Kohlstock[2] have advised for rectal injection the addition of active principles of other laxatives. According to Kohlstock, aloin and cathartic acid, and, in particularly stubborn cases, colocynthin and citrullin should be employed. These minimal enemata are said by Kohlstock to have a prompt and painless effect. But the high price of the remedies unfortunately prohibits their general employment.

The treatment of constipation by means of *subcutaneous injections* has found little favor, but some of the above named remedies and some of the neutral salts (magnesium sulphate) have been employed with more or less success. The same is true of endermatic treatment of constipation by means of croton oil (6 to 10 drops in 15 to 20 grams of olive oil) as advised by Scarbinato.

Furthermore, I must call attention to a mode of treatment of habitual constipation which is apparently paradoxical, nevertheless, in the severest forms, it has produced some surprising results. This is the *rest cure.* I employ this in cases accompanied by general nervous weakness, in which the patient is under-nourished, and in which habitual constipation is the main symptom. In cases which for years had resisted the strongest purgatives, I have seen results in a very short time, usually after even a few days.

The diet is that formerly described. The rest treatment is preferably carried out in a sanatorium, and should be continued for from four to six weeks. Under this treatment the patients usually increase decidedly in weight, which favorably influences the general neurasthenia. By this method, or at least after a very few days, the patient must do without laxatives or enemata, if possible.

Another method of treatment in the neurogenous form of constipation consists in *hypnosis,* which, so far as I know, was first successfully employed by Forel. Lately H. Delius[3] has called attention to the good effects of hypnotism in functional constipation.

From this it is evident that the number of remedies and methods of treatment for habitual constipation is very great, and the list of laxative drugs increases from year to year. In addition, some or even most of these curative methods are used in combination, which makes it difficult to estimate their value.

In my experience the most effectual remedy we possess is a proper diet rigidly adhered to in the manner previously detailed. Occasionally it may be without result, but, in such cases, other curative measures are also fruitless. In addition to the diet for constipation, intrarectal faradization of the intestine alone can be considered a permanently useful remedy, and this is, at the same time, one that can be readily used by the patient. Treatment by enemata and suppositories, as a palliative method, and if correctly used, is harmless in uncomplicated habitual constipation, and, if the other curative measures mentioned fail, this should first be tried. But the other remedies, whether of chemical or physical nature, can be dispensed with today to a much greater extent than is generally supposed. [illegible]

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[1] *Hiller, Zeitschr. f. klin. Medicin,* 1882, IV, p. 481.

[2] *Kohlstock, Charité-Annalen,* 1893, XVII.

[3] *H. Delius, Berliner klin. Wochenschr.,* 1903, Nr. 38.

TREATMENT OF HEMORRHOIDS

[illegible]

struggle between incomplete or immoderate evacuations, both unfavorable conditions for hemorrhoids.

The same may be said of the treatment of hemorrhoids by enemata. Frequently the varices become inflamed by mechanical or chemical irritation, and this increases the sufferings of the patient. At most, mild enemata of oil by means of a soft rectal tube which should be cleansed each time before use may prevent these disagreeable consequences.

In hemorrhoids combined with constipation the rational method consists in producing sufficient and properly formed, i. e., pappy, evacuations by a continuous dietetic treatment, and to prevent fecal impaction in the region of the ampulla recti. As this is made possible in the overwhelming majority of cases, even the very chronic ones, by the previously described diet for constipation, there is, as a rule, no indication for treatment by laxatives. Exceptionally this must be considered, either when rigid adherence to these dietetic rules is impossible or when, in fact, even with careful employment, it produces no result. But in these cases, as already stated, small doses of the mildest laxatives will be sufficient—I allude to the old and reliable compound licorice powder, flowers of sulphur, rhubarb, cascara sagrada, and the like.

Besides the special diet, general hygienic laws, so far as practicable considering the occupation of the patient, must be observed: As much exercise as possible, gymnastics, swimming, rowing, billiards and bowling, lawn tennis and football. On the other hand, it is frequently maintained that horseback riding and bicycling increase the distress from hemorrhoids.

The value of these exercises must not be undervalued, but neither must it be exaggerated; in my experience the latter is most often the case.

According to Oeder,[1] a valuable aid in the treatment of hemorrhoids consists in elevating the buttocks; this may best be accomplished by using a wedge-shaped pillow or by the elevation of the foot of the bed, thus causing a better circulation of the blood from the nodule. This is said to be more beneficial than any form of bodily exercise.

Mineral water cures take a prominent place to-day in the treatment of hemorrhoids and are carried out either at home or at the springs. Here, too, the springs which contain sodium chlorid or Glauber salt are the best. As to the results of this treatment we may refer to what has been previously said, in which perhaps the effect of these cures may be estimated somewhat more highly than in the case of uncomplicated constipation, since here simultaneously the favorable influence of the baths is added. But just as habitual constipation is never cured by mineral water treatment or even relieved for any length of time, neither is there any permanent effect upon the hemorrhoids. It is therefore the duty of the family

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[1] *Oeder, Zeitschr. f. diätet. u. physikal. Therapie,* 1900–1901, IV, Heft 8.

[illegible]

In severe cases it is well not to spend too much time on palliative methods, but to advise the operative removal of the nodules.

Bleeding from hemorrhoids, if only slight or infrequent, requires no special treatment. When it occurs regularly and debilitates the patient, it must be checked.

In such cases we must first attempt to control the hemorrhages by proper regulation of the bowels, i. e., as far as possible to combat the condition by diet. This is by no means always practicable, and therefore to control the hemorrhages, or, at least, to limit them, we must resort to suitable internal or external remedies. For this purpose, styptics have been advised and employed, naturally in the form of anal injections. It is obvious that these remedies (iron chlorid, tannin, alum, lead acetate, ferropyrin, iodoform, etc.) have very little value, inasmuch as they control only a momentary hemorrhage but by no means prevent its repetition. As a matter of fact, I have never seen success from these styptic remedies in cases of obstinate hemorrhage. On the other hand, excellent results are attained by injections of calcium chlorid into the rectum. I order these in a solution of 10 to 100, of which 10 c.c. are injected once or twice daily by means of a small rectal syringe. I am unable to say from my own experience whether the same results may be attained by gelatin injections, but it is quite likely, since the action of gelatin is due to the amount of calcium salt which it contains. At all events, the injection of solutions of calcium chlorid has the advantage of greater simplicity. Even with this method I have occasionally seen failures or imperfect results; but this is exceptional.

Styptics have also been employed internally, and I must mention particularly the extract of hamamelis which is praised by some as almost a specific in the bleeding of hemorrhoids. I have employed the fluid extract of hamamelis in doses of a teaspoonful three times daily, and in some cases unquestionably with good results. To effect a permanent cure, however, it must be continued for many months. Besides the internal use of hamamelis, suppositories of hamamelis (0.25 per dose) may be employed, but I believe that better results are obtained by its internal administration.

In a serious, acute hemorrhage we should not spend much time on uncertain remedies. The most effective is careful tamponing of the rectum with ferropyrin, iron chlorid, or iodoform gauze. Tamponing with gauze dipped in a 30 per cent. solution of hydrogen peroxid is, perhaps, advisable, for H_2O_2, as I have long known, has an excellent styptic action. With this treatment, however, we must be careful to see that the tampon actually reaches the bleeding point, for, otherwise, the hemorrhage will continue in spite of the tampon. At the same time a decided dose of opium should be given internally to arrest intestinal peristalsis. After the hemorrhage has ceased the tampon may be removed, and the intestine may be emptied by oil enemata or the internal administration of castor oil.

Another and very serious complication is *incarceration* of the nodules. Usually the physician is called in after many improper methods to produce taxis have increased the incarceration to the utmost. It is dangerous then to continue efforts to replace these nodules, particularly if they are swollen, of an intense blue color, the surroundings edematous, and the patient moaning with pain. Here quiet waiting is preferable to any active measures. We must be content with elevating the buttocks in the manner described by Oeder, the application of ice to the hemorrhoids, and the employment of opium and belladonna by suppository. If the physician is called to the patient during the daytime, depletion by the application of leeches to the areas surrounding the varices (not these themselves) may rapidly produce amelioration and the desired reposition. If gangrene has already set in, we make no attempt to replace the hemorrhoids, but treat the gangrenous nodules with antiseptic powders (xeroform, airol, vioform and others).

In conclusion, we must mention one of the most disagreeable, although not very painful, complications, *prolapse* of the hemorrhoids. In milder cases, i. e., those in which prolapse occurs only upon defecation, the patients soon learn to aid themselves and readily return the prolapse to the rectum. In advanced cases, prolapse follows any exertion, or, at least, any that is considerable, and then it becomes desirable and necessary to resort to preventive measures. Simple remedies, and also occasionally very useful, are the so-called *hemorrhoidal pessaries* (first advised by Dr. Lütje). But these often fail in their action, causing pressure and pain, and are more often carried in the pocket of the patient than in the rectum.

In severe cases of hemorrhoidal prolapse, a so-called rectal support has been advised by Esmarch, i. e., a sort of plate which is placed over the anus to prevent the prolapse. I do not know whether or not this apparatus has proven useful. I tried it in a few cases of severe prolapse, and it was unsuccessful.

As is shown by the preceding, internal and conservative therapy has a wide field in the treatment of hemorrhoids, particularly if the affection is not too far advanced, and if there are no serious complications; but it also has its limits, when the only relief is by operation. This method will be employed more rarely, the earlier and more carefully we institute treatment by the methods above described.

MUCOUS COLIC AND MEMBRANOUS INTESTINAL CATARRH

By G. HOPPE-SEYLER, Kiel

THE appearance of membranous, tape-like or circular, even net-like masses in the feces has frequently interested the laity and physicians. The occurrence makes the patient anxious, and gives him an idea that these are parasites, tape-worms or the like, or even that portions of the intestine, particles of its mucous membrane covering, are desquamated. If, as frequently happens, there are severe symptoms, attacks of colic or tenesmus, and only very scant, hard masses are voided, among which are profuse amounts of the abnormal particles, it is not necessary that the patient be of a nervous disposition, as most of these patients are, to make him fear that he is suffering from a severe disease. Neither does the physician regard this clinical picture with the easy assurance which he feels, for instance, in the case of an ordinary chronic catarrh of the large intestine which, in consequence of an improper mode of life, has developed into an acute catarrh with frequent relapses. We are not yet certain of the nature of the affection; a positive etiologic and anatomic foundation is lacking, and curative treatment proves to be difficult, tedious and elusive. Scarcely do we think we have mastered the disease when an error in diet, a psychical climax, any irregularity restores it in all its severity. Nevertheless, the medical world has frequently and very actively busied itself with this disease in the last few decades, and the interest which it has awakened finds expression in the great number of designations which have been proposed for it, and which now abound in literature. According to the special view of its nomenclator, the greatest stress is laid either upon the intestinal lesion or upon the nervous disturbances. We find the following terms employed as synonyms: Enteritis s. colitis membranacea, pseudo-membranacea, pellicularis, tubularis, mucomembranosa, crouposa, fibrinosa, etc., pseudoenteritis, catarrhus desquamativus, diarrhœa membranacea, tubularis, etc., finally, colica mucosa, mucous colic, as Nothnagel[1] designates at least some of the cases. Some of these designations apply only to a few of the cases; the designation "crouposa" or "fibrinosa" does not coincide with what we know to-day of the composition of the characteristic membranous masses. As they are principally composed of mucus, nastics or massage. Preferably, laxatives and drastics are taken which powerfully irritate the mucous membrane, and stimulate the muscularis to severe, often spasmodic, contractions. Thus an irritative condition of the mucous membrane develops, which does not go beyond the stage of an ordinary catarrh, but which is combined with a tendency on the part of the intestine to irregular, strong contractions, and upon this foundation of abnormal processes, membranous enteritis readily develops.

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[1] *Nothnagel*, "Beiträge zur Physiologie und Pathologie des Darms," Berlin, 1884; "Erkrankungen des Darms und Peritoneums," Wien, 1895, p. 144.

The disease usually is found in persons of middle life. In advanced age it is rare, and is then the result of a pathologic habit acquired in former years. Not rarely we hear of aged women who had suffered years previously from the passage of membranous masses, and had gradually recovered in later life. In children the affection is rare, yet some undoubted cases are found in literature.

PATHOLOGIC ANATOMY

The *anatomical lesions* in mucous colic and in membranous intestinal catarrh are not yet quite known to us. But few autopsies have been held, and in many of these the question has arisen, in how far the changes found are in actual relation with the affection, and whether they are not to a great extent accidental secondary findings. The disease in itself is not one that endangers life, and therefore is rarely observed at the necropsy; furthermore, we then usually find manifold alterations due to the affection which has caused death. If we omit from consideration the cases not observed during life, the autopsy reports are reduced to merely the cases of O. Rothmann[1] and M. Rothmann.[2] In the patient of the firs author, who suffered from typical mucous colic and who succumbed to a perforating ulcer of the duodenum, nothing was found in the intestine. Th report states: "Although the entire intestinal tract was investigated, th remaining organs showed nothing abnormal." This does not indicat whether or not a histologic examination of the intestinal canal was made In M. Rothmann's case the cause of death was a carcinoma at the bas of the skull. During life there was obstinate constipation; after inte tinal washings numerous strand-like masses of a whitish mucus withou fecal admixture were several times discharged. Pain was not presen In this case distinct changes were found in the mucous membrane, pa ticularly in the descending colon. In the transverse colon were son fecal masses. Where these were absent, the mucous membrane was injecte and formed in folds, and this was particularly the case in the narro contracted descending colon. The narrowed lumen was entirely filled whitish, partly membranous, partly ribbon-like masses without fecal a transparent, as is commonly the case with intestinal mucus. When placed in alcohol the gelatinous masses change to a porcelain white, and become quite tough. They shrink, and do not so readily entwine and dissolve into lamellæ. They may then resemble connective tissue masses originating from food, or fibrinous pseudo-membranes such as are formed in diphtheria.

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[1] *Deutsche med. Wochenschr.*, 1887, p. 602.

[2] *Deutsche med. Wochenschr.*, 1893, Nr. 41.

Upon *microscopic* examination a light, *basic substance* is first noted. This shows manifold fine streaks so arranged that they resemble very thin fibers of connective tissue. On the addition of acetic acid the connective tissue fibrillæ should swell and become indistinct. On the contrary, the striation becomes more distinct and profuse. The striæ form a delicate network, the meshes of which have very sharp contours and are glistening, and include epithelia and round cells. Alcoholic preparations also disclose a coarse, fibrinous basic substance with a deposition of cells, and thus is explained the view of the first investigator of these masses, that they consist of fibrin.

The *epithelia* which are included are usually changed in various ways. Only rarely do they retain their ciliary border; sometimes they are still arranged in rows, therefore detached at one time in large amounts. Some of them still have their normal, cylindrical form; many, however, have enlarged by swelling, and are misshapen, becoming round or more closely resembling pavement epithelium. Others again are elongated and spindle-shaped, so that they resemble smooth muscular fibers, and their terminations are not pointed but rounded off. Fatty degeneration of the protoplasm, the splitting of the cells, as described by Nothnagel, and disintegration up to granular decomposition may be frequently noted; the disintegration of the cells causes them to become friable, and upon pressure on the cover-glass they split up into hard, feebly-glistening, irregular striæ; this process has been referred by Nothnagel to drying, by Kitagawa[1] to hyaline degeneration or coagulation necrosis. Adolf Schmidt,[2] on the contrary, has proved it to be due to imbibition with soaps. Upon addition of caustic potash the cells clear up and the nuclei become more distinct, while an addition of acids does not have this effect. If a particle of the mucus mass is thoroughly admixed with acetic acid or hydrochloric acid, and the preparation is carefully warmed upon an object carrier over a flame, from the cells fine fat globules are apparent which finally collect in large drops under the cover-glass. The cell at the same time clears up, the nucleus becomes distinct; the solubility in ether, and a black staining with hyperosmic acid confirm the fatty nature of the drops which have developed. The cells, in spite of imbibition, are still well retained, for their nuclei stain readily with aniline colors. The epithelia are probably attacked only after desquamation of the fatty infiltration. As the mucous membrane of the large intestine, according to Kobert, secretes fatty acids and calcium salts, and imbibition with calcium soaps, *intra vitam*, would not be impossible, therefore, from what we know of cell alterations of this kind, we can the more readily assume that this process occurs only in the contents of the intestine. The great number of epithelia are indicated by the numbers of those which have a more lamellar character. On the other hand, the solid columnar mucous membrane structures are decidedly deficient in these. The former, to a great extent, are said to be composed of cells, so that intense desquamation of the mucous membrane is to be assumed in their production.

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[1] "Beiträge zur Kenntniss der Enteritis membranacea." *Zeitschr. f. klin. Medicin*, 1890, XVIII, p. 9.

[2] "Ueber Schleim im Stuhlgang." *Zeitschr. f. klin. Medicin*, 1897, XXXII, Heft 3 und 4.

Round cells, in comparison with epithelial, are usually less marked; sometimes they can scarcely be found. A. Schmidt[1] showed them to be more profuse in stained cut sections, and believes they are usually present in larger numbers than is believed, as they frequently disintegrate and in the fresh preparation may therefore be confounded with epithelium. Eosinophiles may also be found among them, with or without the simultaneous appearance of Charcot-Leyden crystals. Plentiful numbers of leukocytes always favor a decided catarrhal or inflammatory process. Masses which consist almost exclusively of pus corpuscles favor ulcerative processes, and in these the mucus intermediary substance is often absent.

Bacteria, which are frequently very numerous in the masses, are of various kinds, and among them are many rods of different shapes, cocci, etc. In many cases hyphomycetes with distinct naked threads are seen. In how far bacteria are the cause of the pathologic process cannot be stated; some authors naturally attribute the disease solely to their presence.

The preparation of dried specimens is not easy, as the smallest particles are difficult to break up; they are not very adherent and constantly slide away; this is probably due to the fat.

Among *crystals* described by Nothnagel are cholesterin plates and thos of ammonia-magnesia phosphate. These, as well as the Charcot-Leyde crystals and the fatty acid needles, are present in the mucus masses i very varying amounts and are by no means constant. Remains of food vegetable as well as animal, are not rarely embedded in the masses.

We arrive at important conclusions by the *chemical examination* o the membranous structures. Naturally this is somewhat difficult, for th masses are but slightly soluble in the mediums ordinarily used for th solution of albumin and mucus products. Cold water will extract som mucin and albuminous products which appear to consist of nucleo-albumi and globulin; these, however, are but small amounts. Weak alkaline s lutions extract but little of mucin and albuminates from the masses stronger solutions will dissolve them, particularly upon heating, but they destroy the mucin, and, therefore, no longer convey a clear picture. As A. Schmidt declared, the difficulty of dissolving the masses which, otherwise, are not present in mucus products, depends upon their very intimate infiltration with fat. If the masses are dissolved by remaining for days in a pepsin solution which contains hydrochloric acid, the fluid becomes turbid, particularly near the surface, and fat globules and fat layers are deposited. If these are shaken up with ether the fluid clears, and upon evaporation of the ethereal solution quantities of fat remain. Schmidt has made some fat estimations. In two cases of membranous enteritis he obtained respectively 19.7 per cent. and 9.5 per cent. of fat from the dried substance. More than one-half of the fat consisted of soap, or fatty acids and soap. In a case of true mucous colic, only 2 per cent. of fat was present. Microscopically, the fat cannot be recognized; no needles of fatty acids or soap are to be seen. Unfortunately, the fat is so difficult to extract from the masses that a solution and investigation of the unchanged mucin is impossible; this is usually altered by extraction. The behavior of the masses in a so-called triacid solution after Ehrlich favors the presence of mucin in abundance. They are stained green, as mucus assumes this color. Fibrin or connective tissue, on the contrary, stains red when brought into contact with this mixture. By the red staining of connective tissue masses originating from the food, of desquamated intestinal mucous membrane, and of fibrinous membranes it is possible to differentiate them macroscopically from mucus masses. Schmidt microscopically determined this in sections of stained and embedded mucous membrane masses, and furnished the proof that fibrin was absent. Only connective tissue remains of the food, which are often intimately admixed with mucus, alone show the stains which have been described. Elastic fibers and the remains of vessels are proof, and will protect us from errors. Acetic acid, as already mentioned, produces in the mucus masses a network of strongly refractive threads, which also is in favor of decided amounts of mucin. Only when the membranes contain much cellular material, and, in consequence, become non-transparent, do they clear upon the introduction of acetic acid, in consequence of swelling of the cell protoplasm. In these cases a larger amount of albumin will be found in the masses, and, upon staining with triacid solution, a more pronounced red color will be produced. In spite of the apparently great masses of mucus which some persons with this affection evacuate daily, they lose comparatively little body-substance. A patient of Schmidt's lost only 0.18 grams of nitrogen in the large quantities of mucus which he voided every two or three days. We cannot, therefore, regard these mucus dejecta as reducing the strength of the patient or as producing any marked change in the organism.

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[1] *Loc. cit.*

Of other symptoms, the *attacks of colic* are the most important; these frequently dominate the entire clinical picture, while the catarrhal symptoms on the part of the intestine are quite secondary. These are cases in which, according to Nothnagel, the disease should be looked upon as a nervous affection, and be designated by the term *mucous colic*. The attacks of colic often come on quite suddenly with severe pain in the epigastric region (transverse colon) or in the left side of the abdomen (descending colon). The patients give us the impression that they are suffering from a very serious disease; they are often feeble, pale, are very anxious, and we might readily think that peritonitis, intestinal obstruction or the like was beginning. But, after a short or longer time, these symptoms disappear, and usually after the discharge of the characteristic mucous masses, often in enormous amounts. The patients rapidly recuperate and no longer complain of anything serious. Obstinate constipation, however, continues, and sooner or later, often even the next day, a renewed paroxysm occurs followed by the same results. Sometimes the attacks of colic occur only once in a few weeks or months, and the affection may consist of only a single attack. If the disease causes only occasional symptoms, the patient in the interval, apart from nervous disturbances which are chiefly hysterical and a tendency to constipation, are in their usual health, and we are justified in designating this affection, after Nothnagel, as mucous colic or *colica mucosa*.

In other cases there is a very profuse discharge of characteristic membranous and ribbon-like masses, *but the attacks of colic are absent*. There is a tendency to constipation, for the relief of which purgatives are often taken, as the patients, usually women, feel better after a thorough evacuation. The use of purgatives, however, appears to aggravate the disease, and to increase the irritability of the wall of the intestine. The constipation in these cases shows the peculiarity that it apparently runs its course without narrowing of the intestine. This narrowing is usually not organic and due to cicatrices or the like, but probably originates in a firm contraction of the musculature of the intestine; to this marked contraction of the intestine we may also ascribe the sensation which the patients have of tenesmus and fulness in the abdomen. They usually declare that they have the distinct sensation of feces being present in the intestine, which they cannot expel. This irritative condition frequently manifests itself by hunger, which, however, is usually soon satisfied. The intake of nourishment is often scanty, as it reflexly produces more decided irritation of the musculature of the intestine and aggravates the discomfort. Eructations are frequent in consequence of involuntary swallowing of air. Many patients resort to purgatives, others administer one enema after another. After the latter, as a rule, nothing is voided immediately; the water is discharged unchanged. Then hard particles of feces appear which in form usually resemble the feces of sheep. Between these masses, or discharged in separate movements, are the characteristic stools. When a large quanty is voided, this often terminates the attack; the patients have a feeling of satisfaction and relief after such purgings, but they are usually weak and debilitated. In these cases of *enteritis membranacea* which run their course without colicky pains, a more or less decided contraction of the large intestine appears to be present. Sometimes with a very flaccid abdominal wall, particularly upon the left side, the organ may be felt as a firm column. But the contraction is not so spasmodic or so excessive as in those cases which are designated mucous colic, and are often attributed to a neurosis of secretion. There are, however, all possible transitional stages from a dull sense of fulness and pressure with a feeling of severe tenesmus and contraction up to sharp and colicky pains. The same conditions are observed in the common diseases of the large intestine, and also after the administration of laxatives. It must be borne in mind that nervous and hysterical patients offer but very slight resistance to such sensations, that their reaction to the irritation of pain is much more ready and much stronger than in normal persons, and we are almost invariably dealing with neuropathic individuals.

If we compare the sensations in the intestine with those which are produced in severe contractions of the muscles of the extremity, where also painful spasms occur (for example, in the muscles of the calves in consequence of over-exertion, circulatory disturbances, etc.), we note that the same causes often produce only a contraction of the muscles without pain. One patient will describe these spasms as very painful, while another reports them as only slight. Therefore, a strict differentiation of the forms of colica mucosa and enteritis membranacea *cannot* be made, even though we recognize the causal influence of the nervous temperament in the well-developed cases of mucous colic, as was first done by Nothnagel. It does not appear likely that the mucus gives rise to the colic. The view appears to me more reasonable that the mucus is profusely permeated with epithelium, and often coats the scybala in a characteristic, firm, membranous mass, and that the irritation of the mucous membrane is due to a catarrh, to irritative contents, and more or less severe contractions of the muscularis, which compress the mucus produced by the epithelial cells, and form it into columns and bands, as has been accurately described by Marchand.[1] At the same time the mucus becomes deficient in water, more compact, and infiltrated with fat which probably originates from the intestinal contents adherent to the surface of the mucous membrane. These masses of feces discharged show the effects of this compression and deficiency of water; they are friable, and in the haustra of the colon are formed into small or large balls. Although other symptoms of catarrh of the large intestine are lacking, nevertheless, the presence of numerous epithelial cells, sometimes also of round cells in the mucus, is in favor of catarrh, and the production of mucus is explained, as well as the irritation of the intestinal wall which leads to colic and to spastic constipation; and these are much more fully and more naturally explained than by the very problematic hypothesis of a mucus neurosis of secretion. That the affection generally occurs in nervous, hysterical women naturally favors the view that abnormalities of innervation here play a part. If, however, there is a more decided irritation of the intestinal nerves, this generally expresses itself in the muscular functions of the intestinal wall. In excitement, in fright, under psychical emotion, as is well known, spastic contraction of the intestine or increased peristalsis readily occurs, and this is particularly marked in chronic catarrh. Here, in excitable persons, spastic contraction of the large intestine and ensuing constipation arise, which are readily relieved by the administration of preparations of belladonna, etc. In the disease under consideration we are dealing usually with easily excited, sensitive persons, in whom an irritative condition of the intestine very readily expresses itself by spasm. If this occur in the intestine, it is followed by a narrow constriction and very decided pressure of the mucous membrane which is now stimulated to a profuse secretion of mucus. On account of a lack of reliable reports of anatomical investigations, and the difficulty involved in the observation of the intestine during the attack, it is as yet impossible to explain with certainty the nature of the affection; but I believe that the above solves the question of the origin of the characteristic mucus formation, and that the other symptoms of the disease may be brought into relation with this without accepting a doubtful hypothesis.

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[1] *Berliner klin. Wochenschr.*, 1875.

PROGNOSIS

In regard to life the prognosis is generally favorable; but the disease may lead to a lowered state of nutrition and loss of strength, and thus result in chronic invalidism. As a rule, however, the patient attains old age, and later gradually outgrows the affection. The disease may also be cured by proper treatment, combined with good nutrition and the regulation of the mode of life; usually, however, there is a tendency to relapses. In some cases the disease exhausts itself in a few paroxysms, and, in the first attacks, this leads us to hope that they may be the only ones; decided increase in weight, improvement of the general condition, and the disappearance of other nervous disturbances enhance the hope of cure.

DIAGNOSIS

The diagnosis is easy when we find in the dejecta the typical, membranous, ribbon-shaped, net-like or columnar mucus structures. In nervous persons who now and then suffer from severe attacks of colic, from tenesmus and constipation, it is well to examine the feces, and to ask·

the patient, or those about him, to observe whether masses of this kind are discharged. Cases of constipation and intestinal catarrh which run their course with colic will then be recognized as intestinal colic or membranous catarrh. It is true we must guard against error in confounding these masses with particles of connective tissue such as originate from meat and other constituents of the food. Frequently, connective tissue masses containing globules of fat which, by the effect of digestive juices, have become saponified and have therefore a whitish color, will be taken for membranous mucus masses. I have often seen them after the eating of raw ham, and they have been shown me by anxious patients or their physicians. They closely resemble the mole-like mass which Rosenheim depicts in his text-book in the chapter devoted to enteritis. By their tenacity when we attempt to separate these masses and entwine them, and the microscopic evidence of particles of transverse muscle between the fibers, and the clumps of whitish, fatty tissue, we may easily differentiate them; moreover, they do not show the reaction from mucus mentioned above, nor do they present coarser connective tissue fibrillæ with remains of vessels, elastic fibers, etc.

Besides tendons and fascia, the lumen of arteries and veins, remains of oranges, milk coagula, masses of fungi having membranes, such as the oïdium, may be confounded with mucus dejecta, but the chemical and microscopical investigation of these objects will enlighten us.

The typical mucus masses are disentangled upon shaking in water and are dissolved into countless small particles, which, upon standing, again separate from the turbid fluid and collect upon the surface. This does not happen with the particles of food, etc., referred to above.

Admixture of blood in the mucus masses favors ulcerative processes; therefore, either dysentery or tuberculous disease of the intestine, deeply invading follicular ulceration or ameba enteritis may be simulated.

TREATMENT

The treatment must be designed to combat the various factors which have been operative in the development of the disease, and which are the individual components of the clinical picture. It must therefore control the irritation of the intestinal mucous membrane, the motor intestinal disturbance, and, particularly, the general changes in the nervous system, the hysterical, neurasthenic and hypochondriacal symptoms. This may be attained by careful regulation of the diet, for, on close investigation, we usually find here much at fault! Often the patients, from a fear that certain food will disagree, limit their diet too much. Mechanical and drug treatment of constipation must also be instituted. All irritation of the intestine should be avoided, and when there is decided evidence of bacterial decomposition of the intestinal contents, the administration of disinfectants is indicated. All means to regulate and strengthen the nervous system are, in general, to be employed to the fullest extent.

As the clinical picture varies greatly in the individual patient, at one time this, at another time that, symptom becoming more prominent, special attention must be given to the intestinal catarrh and the accompanying constipation; in other cases, however, in which the catarrhal symptoms are less marked, and a more or less purely nervous affection, therefore Nothnagel's mucous colic, appears to be present, the main stress must be laid upon the regulation of the mode of life so as to avoid deleterious effects which might react upon the central nervous system,—the psychical element. All means must be employed to strengthen the tone of the nervous system.

If examination reveals a well-developed *catarrh of the large intestine*, this must first be treated, primarily by *laxatives*, such as castor oil, rhubarb and the like, thoroughly to cleanse the intestine of long-retained, irritating masses, the generators of fermentation and decomposition. For the latter calomel, a single dose of 0.3 0.5 grams, or, upon continuance of the bactericidal action, the dose repeated for several days, or a dos three times daily of 0.05 0.2 grams will be serviceable. To empty th intestine we employ purgatives, and we assist their action by the use o injections in a manner to be later described; the laxatives render the ir testinal contents fluid and propel them, but, frequently, in the lower pa of the intestine, this effect is lost, and the fecal masses collect here an are not discharged. The prolonged use of purgatives and also miner waters, such as the purgative water and springs containing Glauber's sal is not advisable, as these often have a most unfavorable effect upon t motor irritation and, in general, upon the nervous condition. Therefor a treatment at Carlsbad, Marienbad, etc., or at the saline springs, such Homburg and Kissingen, is generally not so beneficial as in a simp chronic catarrh of the large intestine.

Sometimes the use of *astringents*, bismuth subnitrate or bismuth s icylate, and in cases of decided flatulence magnesia, is also of value. Th remedies may be combined, or an extract of hyoscyamus or oil of pepp mint and the like may be included to allay intestinal irritation.

In the removal of the *motor disturbances* in the intestine, careful m sage will be found serviceable in the interval between the attacks, and t application of the faradic or galvanic current, cautious gymnastic ex cises for the trunk and leg, moderate physical exercise, such as walki horseback riding, bicycling, to combat the frequently coexisting atony the intestine. In such instances laxatives should be avoided as much possible, *enemata* taking their place, and where there is decided irritabili of the intestines, warm injections of Ems water, or a $\frac{3}{4}$ per cent. sali solution in quantities up to one liter. Previous to this olive oil may injected, provided the intestine is but slightly sensitive. Enemata of $\frac{1}{2}$

liter of oil, according to the methods of Kussmaul and Fleiner, are also valuable. The addition of irritating astringents or disinfectants, such as silver nitrate, tannin, corrosive sublimate and the like, had better be avoided. Drugs which have an anodyne effect, as oil of peppermint, starch, etc., may be added to the injections of water, or the injections may be of chamomile or peppermint tea. These intestinal washings act especially well during the attack; they assist in discharging the hard scybala and masses of mucus, as well as the flatus which inconveniences the patient greatly, and thus serve to quiet the intestine. In the interval between the attacks, preparations of strychnin are best employed, such as the tincture of nux vomica with tincture of rhubarb, which mildly stimulate the intestinal movements; during the attack of colic the belladonna preparations, such as extract of belladonna, in doses of 0.02–0.04, or atropin sulphate, etc., afford relief. Even where the spasmodic contractions of the intestine are painless, these drugs are to be recommended. Morphin and opium should only be employed in cases of necessity. To allay the spasmodic pain, heat may be applied in the form of cataplasms, such as may be combined with the use of the Quincke heat apparatus, or as hot woolen cloths, thermophores, etc.

In cases of a more catarrhal character, therefore, in enteritis membranacea, the *diet* should be non-irritating, and acids, spices, fats, foods rich in cellulose, and those which ferment readily, should be avoided. But this restriction in diet must be only until the marked catarrhal symptoms, such as periodically appearing diarrhea, fermentation, and profuse gas formation, are removed. As a rule, a strengthening and nutritious mixed diet is more stimulating and serviceable in the treatment. A continuous filling of the large intestine with cellulose and other remains of food decidedly stimulates peristalsis without leading to spasm, which readily occurs in an empty intestine on account of the difficulty of propelling the compact mucus masses. For this reason plentiful amounts of bread are usually ordered, not fine wheat bread but the coarser varieties which must be well baked; vegetables rich in cellulose, as well as berries and fruits; meats which contain fat, also bacon and butter. Patients are usually anxious to avoid such a diet, for they believe their intestines to be much too sensitive to retain this food, and, if a few attacks recur, it requires great powers of persuasion to induce them to resume this diet. All strongly spiced food, as well as cabbage and other vegetables which are apt to generate quantities of gas, must be excluded, for this accumulation of gas in the intestine causes great distress to the patient, and readily induces colic; the plentiful ingestion of fat and carbohydrates has the further advantage of increasing the accumulation of fat in the body, which should be particularly borne in mind in the case of nervous, thin individuals who have a downward displacement of the stomach, intestine, kidney, etc.; that is, when enteroptosis is present. The fact that in this condition the organs are better supported, that the large intestine has a firmer hold upon the mesentery and omentum rich in fat, that torsion of the organs and their nerves is lessened, and movements of the colon are increased conduces to improvement. In cases where the marked effect of lacing exists, proper corsets should be worn and the dress so arranged that the weight of the clothes will rest upon the shoulders rather than upon the abdomen. In cases of pendulous abdomen with marked flaccidity of the abdominal walls after pregnancy, or from a previous abdominal tumor, etc., the wearing of an elastic abdominal bandage is advisable, prolonged standing or sitting is to be avoided, and there should be frequent rest in the recumbent posture.

In controlling the *nervous symptoms*, general rules must be enforced which cannot here be described in detail. This necessitates careful attention to the complaints of the patient, an accurate study of his mode of life, his manner of thought, and the possible unfavorable factors in his occupation, his family relations, etc., which excite and disquiet the psychical sphere, shock the nervous system, reduce the normal resistance to pain and unpleasant sensations, and frequently lead to nervous tension. Sufficient rest at night, occasional interruption of the day's activity by intervals of rest, and especially a certain freedom in diet must be insisted upon. Detailed hygienic instruction by the physician will often relieve these conditions. In cases of necessity, change of climate, residence in a quiet place in the mountains or at the seashore, will be serviceable. Exciting life at summer resorts or at the seaside is to be avoided. These measures may be assisted by carefully chosen local hydriatic procedures, such as cold rubbings, baths, or douches, general massage and faradization, to the abdomen. Finally, as previously mentioned, a generous diet consisting of fat and carbohydrates will be useful in promoting the accumulation of fat in the body.

In general, the treatment of enteritis membranacea and mucous coli is not easy; but a careful study of the intestinal and nervous disturbance present as well as of the circumstances of the patient, and the investigatio of all the varying symptoms, may enable us in many cases, with the patien help of the sufferer, to cure the affection, or at least to alleviate it, so tha he has intervals of relief from the affection, and it does not so decidedl interfere with his occupation.

INTESTINAL CONSTRICTION AND INTESTINAL OCCLUSION

BY H. NOTHNAGEL, VIENNA

I. INTESTINAL CONSTRICTION

THE two clinical histories which we shall here analyze and discuss belong to the severest forms of disease with which the physician is brought into contact; chronic constriction of the intestine in the one case, and acute occlusion in the other. I shall detail the pathology of these two conditions as definitely as brevity will permit, and shall first describe stenosis, then intestinal occlusion, proceeding from the simple to the more complex.

The first patient, a woman aged 37, states that she has had five normal labors, and, with the exception of an acute febrile articular rheumatism in her eighteenth year from which she recovered, has always been well. Three months ago she began to suffer from constipation, while previously the bowels were normal. This symptom developed quite insidiously without any preceding change in the mode of life of the patient. She was compelled to take purgatives, which formerly had never been necessary. In addition there was pain of a colicky character, which occurred daily, never lasted more than a few minutes, and frequently subsided with the passage of flatus. Moreover, the patient had observed that, for about five weeks, during the attacks of pain, the intestines and different parts of the abdomen bulged and formed prominences (as she expressed it). In the last three months she has emaciated appreciably.

The totality and grouping of these symptoms are characteristic, and must at once arouse a suspicion of intestinal narrowing. Constipation developing insidiously without recognizable external cause, frequent pain of a colicky nature, and, above all, the bulging coils of intestines, form the characteristic features. The symptomatology of intestinal constriction is simple and comprehensive: The clinical picture is dominated by the fact that the propulsion of the contents of the intestine meets resistance at a certain point due to some anatomical obstruction.

SYMPTOMATOLOGY

Chief among the *symptoms* are *alterations in the fecal movements.* Constipation develops slowly and gradually, since the affection which causes the stenosis also almost invariably arises insidiously. This kind of constipation is important in diagnosis for the reason that it occurs in persons who have previously had regular evacuations, and no other cause can be assigned for its presence. At the onset, according to the situation of the stenosis, mild therapeutic measures are sufficient for its amelioration—purgatives by the mouth or irrigations per rectum; gradually, however, other symptoms appear while evacuation of the intestines becomes more and more difficult.

The degree of intestinal activity is pre-eminently influenced by the seat of the stenosis; therefore, while the contents are still fluid or semisolid in the jejunum and ileum their passage is not greatly interfered with, or, at least, only late in the course of the affection; on the other hand, this is marked in the lower portions of the large intestine. It happens occasionally in stenosis of the colon, even in advanced stricture of the sigmoid flexure, that the daily evacuations continue for a long time to be regular.

Occasionally, constipation is interrupted by the appearance of diarrheal discharges, and then enormous masses may be passed. This diarrhea is the consequence of irritation of the walls of the intestine above the stenosis by the retained contents and may persist for a long time. Blood and pus are sometimes admixed with the dejecta, originating, in rare instances, in a secondary decubital ulcerative change in the mucous membrane above the stenosis, more frequently due to definite anatomic forms of stenosis, particularly carcinomatous changes. Special importance in diagnosis is often attributed to changes in the shape of the fecal masses (*feces of stenosis*) : small balls resembling sheep feces, containing one or two grooves; or thin, lead-pencil-like, or flattened ribbon-like, columns of feces. Formations of this nature are not often observed, but, in such cases, they cause confusion for they may occur without any anatomic change in nervous spastic constipation or in cases of so-called starvation intestine.

These changes in the feces, considered individually, are of slight diagnostic value, and this is also true of the other symptom which is sooner or later added, namely, the *colicky* attacks of *pain*. The intensity of these pains varies greatly; sometimes they are extraordinarily severe. In character they are like other paroxysms of intestinal colic. Their seat is generally the region of the stenosis, and from this the pain radiates to other areas of the abdomen; rarely are they more severe anywhere than at the point of their origin. Gurgling, rumbling sounds are often noticed during the paroxysms. Their physiologic foundation is the increased resistance to the propulsion of the intestinal contents, and the decided intestinal contractions thereby produced.

One of the most significant symptoms, often decisive in diagnosis and occasionally pathognomonic, is the *plastic prominence of the tetanically dilated, tense coils of intestines,* with or without visibly increased peristalsis. This tetanic, visible, and palpable phenomenon, designated by me as *intestinal stiffening*, finds conditions for its development only in the presence of intestinal stenosis, with accumulation of intestinal contents in a gaseous or fluid form, with energetic contraction of the intestine as a result of hypertrophy of its walls, and resistance to the propulsion of the intestinal contents. We distinctly see and feel how a portion of the intestine (perhaps already distended with gas but still soft) is changed, usually quite suddenly, into a ridge standing out prominently and rigidly above the surrounding area and actually to be grasped by the fingers, which remains in this condition for a short time, and then returns to its former elastic state. After a few minutes this is repeated. The patient suffers severe colicky pain which is produced by the tetanic condition of the intestines. For the diagnostic employment of this phenomenon, it must be shown that tetany does not occur in an empty coil, or one that can be emptied, but in a dilated coil, one filled with gas and intestinal contents, which, on account of the obstruction (stenosis or occlusion), cannot relieve itself.

Besides intestinal stiffening, *increased peristalsis* is often present, and is manifested by a tortuous, more or less active forward motion, visible over limited or extensive areas.

The symptom just mentioned (with changeable conditions) is characteristic of intestinal constriction; all other symptoms occurring in a concrete case are incidental consequences, or due to the nature and anatomical composition of the stenosis. The external condition of the abdomen varies as much as the results of percussion; both are determined by the fulness of the intestines, be it with gaseous or compact or fluid contents, which vary from day to day or from hour to hour. General diagnostic rules cannot be formulated. One condition must be briefly described; occasionally the fluid mass which has accumulated above the stenosis becomes so massive that the greatly dilated coils (on account of their weight) drop from their horizontal position to the sides of the abdomen, thereby simulating ascites, especially since, with this change in position, the full coils of intestines may also change their location. I have observed in stenoses of the sigmoid flexure as well as of the ileum that coils of the small and also of the large intestine may fall into the flanks. A very simple maneuver will decide in such cases whether ascites or the fluid contents are present in the lumen of the small intestine. If a few sharp taps are made longitudinally upon this area, dull upon percussion, and a splashing sound is produced, it is certain that fluid is inside the lumen of the intestine.

In the case whose history has just been detailed the characteristic symptoms were present and the diagnosis of enterostenosis could be made with absolute certainty, but this is not always the case. The diagnosis is sometimes simply impossible, if no obstruction can be felt, and when the contents of the intestines are still expelled, either because very fluid, as in the small intestine, or because the obstruction is not yet sufficiently advanced. Between these extremes is a series of cases which seem more or less probable, but which necessitate great care in the diagnosis. A positive decision in a given case depends upon two things: either we must feel the stenosis directly in the rectum—that is, we must palpate a tumor, a peritoneal band—or, upon the surface of the intestines, we must detect a special configuration, the tetanic intestinal rigidity and increased peristalsis during an attack of colic.

What lends to the rigid, chronic stiffening of dilated coils its great diagnostic value, particularly for stenosis? The anatomical change above the narrow area furnishes the explanation of this. The intestine is here dilated, usually only to a limited extent and moderate degree, but sometimes over large areas until an extreme degree is reached. Macroscopically, the distinctly visible muscularis of the intestine is hypertrophic, in cases that are prolonged often decided, increased over great areas, for instance, in stenosis of the sigmoid flexure, up to the ileo-cecal valve. This thickening of the intestinal wall, which in acute stenosis may even be recognized upon the fourth or fifth day, is the product of a true hypertrophy of individual fibers and muscles which owe their origin to increased muscular exertion that is absolutely necessary to propel the intestinal contents through the narrowed area. And as such a marked muscular hypertrophy, capable of developing an enormous rigid stiffening, only arises in extreme stenosis, the diagnostic importance of intestinal rigidity is clear.

ETIOLOGY

We now turn to the question: What etiologic factors are capable of producing such a change in the intestinal lumen that the propulsion of the contents becomes difficult? This condition we designate as constriction of the intestines (stenosis) in contrast to intestinal obstruction (occlusion) in which the lumen is completely closed, and any propulsion o the contents downward, i. e., in a physiologic direction, is absolutel impossible.

*The form of, and the causes which may produce, **narrowing** of th lumen of the intestine, are manifold.*

We should digress far beyond the limits of this article if we attempte minutely to investigate these, and must content ourselves with their simp enumeration, giving to each the brief consideration necessary for o concrete purpose.

First, it must be stated that the condition designated as intestin stenosis is, in a clinical sense, always due to anatomical changes and rel tions. Certainly, a spastic contraction of the musculature of the intestin will cause a narrowing of its lumen. This, however, is but a tempora condition, and does not produce the clinical picture characterized by the term "intestinal stenosis." The fecal masses which accumulate in the intestine, especially in habitual constipation, also narrow the intestinal passage; but in this condition the picture of intestinal stenosis with muscular hypertrophy and tetanic stiffening does not develop; should any serious local consequences arise, the symptom-complex of complete intestinal occlusion results.

Chief among the causes of stenosis of the intestine are the following:

1. *Carcinomata of the intestine.* These are almost always primary; only very exceptionally does a carcinoma develop in the intestines by way of metastases, through the blood, or through the lymph-vessels. Naturally we must consider very differently those not infrequent cases in which the neoplasm proliferates from a neighboring organ (the stomach, the uterus, the gall-bladder) to the intestines.

Universal experience teaches us that carcinoma in the small intestine is very rare, is frequent in the colon, and most frequent in the rectum. To illustrate, we will mention only those figures which are taken from the autopsy reports of the Pathological Institute of the Vienna General Hospital. In twenty-four years, among 41,838 autopsies there were 343 intestinal cancers. Of these 7 were in the duodenum, 10 in the ileum (none in the jejunum), 164 in the colon, and 162 in the rectum. Accordingly—and other autopsy statistics agree in this—the number of colon and rectal carcinomata is almost equal. In contrast to this, the statistics in the living show the very extraordinary preponderance of rectal cancers, 80 per cent. of all intestinal cancers being here included. The discrepancy in these statistics may probably be largely explained by the fact that cancer of the rectum is much more susceptible to diagnosis and more readily operable, so that many carcinomata of the rectum are operated upon, and do not come to autopsy in the Hospital.

Clinically, the fact is noteworthy that cancer of the intestine occurs with relative frequency in the first half of life, usually developing before the fortieth year. The anatomical peculiarity of intestinal cancer, that it often distributes itself in a markedly annular form, occasionally encircling the intestinal lumen like a cicatricial ring, makes it possible for the neoplasm to produce decided narrowing which may be so extreme that only a thin pencil or a sound can be introduced. The same stenotic effect, however, may occasionally be produced by all other forms of the neoplasm.

2. *Sarcomata* may also narrow the lumen, but this effect is only rarely observed. On the contrary, in this form of tumor, the intestinal passage is but little influenced; on account of the peculiar distribution of the neoplasm mass into the musculature, the intestinal wall rather becomes paretic.

3. I include here *tuberculomata* of the intestinal walls, which, like car-

cinomata, and in identically the same manner, may bring about stenosis. I have observed several cases of this form of tumor and the literature of the past year also furnishes examples.

4. *Benign intestinal tumors* (myomata, lipomata, angiomata, fibromata, adenomata) only exceptionally form the cause of stenosis; their entire composition and anatomical formation is but little conducive to this and their occurrence is quite rare. At the most they indirectly cause a limitation in the passage by becoming the starting point of an invagination as pediculated polypi. The majority of benign tumors which, by direct limitation of space, cause stenosis are situated in the rectum and usually occur in children.

5. *Internal cicatricial strictures* are produced from connective tissue, from cicatrices of ulcers. Of the many varying etiologic forms of ulcer in the intestine, few terminate in a cicatrix which causes a stricture. The most frequent are the tuberculous and stercoraceous ulcerations, more rarely the round ulcer of the duodenum, the dysenteric and catarrhal follicular ulcerations, the syphilitic forms of disease, finally, traumatic lesions and enteric fever ulcerations. This scale of frequency is different from that commonly given, in which the dysenteric cicatricial ulcers occupy the first place, and the tuberculous and typhoid ulcers the last. According to the comprehensive statistics compiled by Woodward, we can hardly doubt the rarity of dysenteric strictures. On the other hand, König has called attention to the fact, opposed to former views, that tuberculous cicatricial strictures have been frequently observed, and this coincides with my own personal experience.

Tuberculous cicatricial strictures occur mostly in the small intestine, particularly in the ileum, as do also the very rare typhoid strictures; those due to ulcer of the duodenum are naturally found in its region; dysenteric and stercoraceous (decubital), in the colon and rectum. The cicatricial stenoses after catarrhal follicular ulcers, which are also exceedingly uncommon, are met with in the small as well as in the large intestine; those due to syphilis preponderate in the large intestine, more accurately, in the rectum. This latter region, more often than any other part of the intestine, may become stenosed by cicatrices which originate from trauma,—operations for prolapse of the rectum, destruction of hemorrhoidal nodules, injury from syringes or introduced foreign bodies, and to these perforating periproctitic abscesses are superadded. Cases of stenosis of the intestine are rarely observed, and up to this time have been exclusively in the small intestine, after severe trauma of the abdomen which has produced a lesion in the wall of the intestine. These anomalies have a purely clinical interest from the fact that when a portion of the intestine is implicated in a strangulation, or after the desquamation of an intussuscepted portion of the intestine in invagination, a stricture may subsequently develop.

6. *External constriction of the intestine by peritoneal processes*, the anatomical appearance of which has been portrayed by Treves. It is impossible for us here to give a detailed description; only the most important general features will be emphasized. In this etiologic group we invariably deal with chronic peritoneal adhesions, indurations or bands, which have made a portion of the intestine adherent either to another part of the intestine, to any area of the abdominal walls, or to another organ, and, in the latter case, to one normally in the neighborhood of the intestine or abnormally situated there.

In very different ways the lumen of the intestine may be occluded by the action of chronic peritonitis. Peritoneal indurations which are isolated and circumscribed usually encircle the intestine in a ring-shape, or, at a circumscribed point on the lateral surface, the adherent intestine becomes more or less decidedly twisted, when we have a volvulus of the intestine. Or, a general peritonitis leads to concretions, partly of the intestinal coils among each other, and partly of the parietal serosa with the solid abdominal organs. Occasionally, constriction occurs as the result of peculiar extension of the intestinal wall by traction of a diverticulum.

I must refrain from the enumeration of the various anatomical causes and the etiologic modes of development of this chronic adhesive peritonitis, and shall only state that this etiologic form of stenosis may affect the different portions of the intestine quite irregularly.

7. *The intestine may be constricted by a tumor or by any other mass acting similarly by pressure* (gravid uterus, dislocated organs, inflammatory abscesses). In this group those portions of the intestine are most frequently occluded which, on account of their anatomical position, and their tense, mesenteric fixation, are least able to elude pressure; first, the rectum, then the descending colon and the sigmoid flexure, the duodenum, the ileum infimum, the upper portions of the ileum very slightly, the jejunum and the transverse colon.

Besides the factors which have been mentioned, in very rare cases other conditions may bring about a constriction of the lumen of the intestine, such as a chronic invagination, or even sometimes an obstruction from hard fecal masses, gall-stones, or a foreign body. But this is exceptional; for the most part the conditions enumerated are the causative ones.

We now return to our special case with the question: What causes the stenosis in this patient? In which portion of the intestine is it situated? I will recapitulate briefly the important results of our investigation: The patient is afebrile; moderately emaciated; there is pallor of the face; the radial pulse is normal; there is constipation and the previously mentioned abdominal pain. Upon inspection the upper portion of the abdomen shows nothing abnormal. It is situated below the level of the thorax; the lower half, from the navel downwards, is somewhat inflated; there is no meteoric bulging of the flanks. In the lower half of the abdomen, occasional tetanic intestinal rigidity and progressively energetic are noted. Most decided bulging, in extent that of a dinn periodically observed in the ileo-cecal region. This area is als of the severest pain. These phenomena appear spontaneously, ca which ceases after a few seconds, at most after a few minutes. ally loud gurgling and rumbling are heard. Stress must be laid following: If the abdominal surface is perfectly quiet, it is possible to produce peristalsis and intestinal tetany by gentle but, more certainly, by short, energetic taps which invade dee flagellations with a cold, damp towel.

The seat and the relief-like arrangement of these characteris must be accurately studied; as their presence permits the diag intestinal stenosis, their seat and arrangement furnish valuable a rule, the most important aid in answering the question in wh of the intestine the stenosis is situated. I go still further sinc the average, the determination of the seat in chronic stenosis of th tine is easier than giving an opinion in regard to its anatomical as is shown by the above compilation of the anatomico-etiological tions, certain processes which cause stenosis occur very frequently i vidual portions of the intestine, in others very rarely or not at al by determining the seat we indirectly gain, at the same time, points of support for the recognition of the anatomical process. F *reason, I generally advise that we first, if possible, determine the the obstruction, and then its nature.*

Descriptions of the various forms of intestinal stiffening and peris which occur in individual cases are hardly possible; illustrations a much clearer idea of the condition. I shall, therefore, limit mysel the enunciation of a few general principles.

As a rule, the arrangement and position of the intestinal areas corres with the normal.

Distention of the flanks and of the upper abdominal regions prima points to the colon. If the flanks are not at all distended, if they f a horizontal line, or are even concave, stenosis of the colon may, great probability, be excluded; but the possibility of congenital or acqu anomaly of position must always be considered. The inverse, howeve not true; i. e., that extension of the flanks may be caused by tensely f and prolapsing coils of the small intestine. The diagnostic importanc extension of the flanks is especially convincing when the obstructio situated in that portion of the colon from the hepatic flexure to the sple flexure; here the right flank alone is distended. In the majority of ca of chronic stenosis, the dilated, stiffened or extremely peristaltic lar intestine is in its normal situation (while the enormous degree of infl tion in acute obstruction, especially in sigmoid volvulus, creates the mo remarkable topographical positions).

Abnormally situated intestinal coils, with alternating and disappearing stalsis or stiffening, indicate the small intestine. The same is true everal (2, 3 or more) adjacent, oblique or vertical (organ-like) coils n the longitudinal axis of the body.

The most extreme thickening, as a rule, must be referred to the intes- l portion situated nearest the obstruction, which is accompanied by ked muscular hypertrophy. An exception to this occurs only when intestine has become paretic by long-continued over-distention.

The fact is noteworthy that movements in the colon are usually slow, he small intestine they are more rapid and irregular. It is often noted t they cease in a definite locality, with absolute regularity, for example, the ileo-cecal region, and if there is irregular peristalsis of the small estine, if the lateral walls invariably remain quiet, we conclude that obstruction is in the region of the cecum.

These relief-like circumscribed inflations and changes in the shape of abdominal surface may, however, be entirely absent. The seat of the nosis is then much more difficult to decide, but it may occasionally made with certainty, provided physical examination reveals such rela- ns as are calculated to influence directly or indirectly the permeability the intestine. To this category belong *the proof of tumors* of a neo- asmatic or inflammatory nature, changes in position of the solid abdom- al organs, tumors from invagination, chronic peritoneal bands, and old ternal hernias.

The results of percussion and auscultation are less valuable in deciding pon the seat, are only to be utilized most cautiously, and then rather cases of occlusion; in stenosis little or no value can be attached to this ethod of examination.

Vaginal exploration is an important aid, and even of greater signifi- nce is *digital rectal examination,* by which we may directly demonstrate e obstruction which produces the stenosis. These examinations should ever, under any circumstances, be neglected, for with the regular *exam- ation of the hernial rings* they become a duty upon which too great ress cannot be laid.

Pain is a very unreliable symptom. Only when it is closely circum- ribed, accurately located, of uniform degree, and permanent, can we trace s seat to a definite locality, and not always then.

The stenoses of the duodenum and the rectum call for special dis- ussion.

Chronic stenosis of the duodenum gives rise to varying symptoms, ccording to whether the narrowed area is situated above or below the ommon gall-duct and the pancreatic duct. The clinical picture in the ormer case so closely resembles that of stenosis of the pylorus that a ifferential diagnosis is impossible. Where the constriction is deeply situ- ted there is also gastric dilatation; in this case, however, there are characteristic symptoms in addition. Bile flows profusely and continuously into the stomach so that massive bilious vomiting occurs; or, after gastric lavage, large quantities of pure bile may flow from the tube. Pancreatic juice may also be present in the stomach, so that in this organ duodenal digestion with distinct splitting of fat may take place.

Stenosis of the rectum is easily and most positively diagnosticated, as, in the majority of cases, it may be reached by the palpating finger. Sometimes suspicion is awakened by the apparently causeless development of hemorrhoids and hemorrhages; also by tenesmus. In the main, general diagnostic laws are here applicable.

The employment in our special case of the rules which have been briefly sketched forces us to the view that the seat of the stenosis is in the lowest part of the ileum, in the neighborhood of the ileo-cecal valve (the details of proof may be omitted, and we may assume the diagnosis to be correct since later, when laparotomy was performed, this view was confirmed).

Another question arises: *What is the nature, what the anatomical composition, of the obstruction which causes this stenosis?* The determination of the anatomical nature of the intestinal obstruction is hedged in by enormous difficulty, and, in our opinion, forms one of the most perplexing points in diagnosis. Only the most careful consideration of each individual symptom enables us to proceed, and we should never forget that each concrete case may deviate from the symptomatologic type.

The general rules must be first mentioned.

Primarily, the hernial rings should be examined. Hernias are not only of great importance in acute intestinal occlusion, but old hernias may also form a focus for chronic constriction by means of peritoneal adhesions which lead to constriction and volvulus of the intestine.

Exact digital investigation of the rectum and vagina must be made; this occasionally furnishes the direct clue to the diagnosis, or points the way to it.

If examination of the hernial rings, of the rectum and vagina, is negative, the abdomen must be carefully palpated. In a certain number of cases a tumor will be found, and we must decide whether it be a malignant growth or chronic invagination, a fecal mass or an inflammatory infiltration. The points of support operative here cannot be described. Naturally tumors, particularly those not circumscribed, may be present and escape detection, either because they are too small (which is particularly the case in carcinoma), or because they are concealed by inflated coils of intestine, or for other accidental reason.

Occasionally we succeed in palpating isolated, thicker, peritoneal bands of irregular contour. If we are dealing with tough, massive, distributed and chronic, inflammatory adhesions, such, for example, as are produced by tubercular peritonitis, the anatomical situation clears at once.

If, however, no clue is found which may directly lead to the recognition of the anatomical nature of the obstruction, then an explanation must be sought in the history, in the course of development, and from other circumstances. Chronic inflammatory processes in the pelvis, a preceding perityphlitis, and similar anamnestic data indicate peritoneal fixation, dysenteric and other ulcerative intestinal processes point to cicatricial strictures, very insidious development of the symptoms of stenosis without preceding disease indicates carcinoma. Under these circumstances, it is of special importance in the diagnosis to determine in which portion of the intestine is the narrowed area.

Let us apply this mode of investigation to our special case.

The hernial rings are free. Palpation of the abdomen reveals the intestinal rigidity and increased peristalsis which have been described, which justify the conclusion that the seat of the obstruction is the lowest portion of the ileum—but, beyond this, absolutely nothing to assist in explaining the nature of the obstruction, no palpable tumor or peritoneal band, not even an area painful upon pressure. On the other hand, vaginal and rectal examinations yield positive findings: In the left vaginal vault, a prominent crescentic cicatrix, on the right side a narrow cicatricial band extending to the posterior wall of the vagina. These cicatricial adhesions render a positive palpation of the adnexa difficult. Palpation by rectum shows the pelvis to be filled with markedly inflated intestinal coils, and, besides, particularly upon the right posterior surface of the uterus, posteriorly toward the ligamentum latum, thin, tendinous, coarse bands of adhesions may be distinctly felt. A *preceding pelvic peritonitis* must, therefore, be diagnosticated.

On account of these conditions, the opinion naturally forces itself upon us that these chronic peritoneal processes arising from the internal genitalia are connected with intestinal stenosis, the latter being due to constriction or volvulus. In connection with the otherwise entirely negative findings, this conclusion must be recognized as logical, and no direct, forcible objection to it can be raised.

Nevertheless, we must be careful not to assume without more ado that this conclusion is correct. This caution is not based upon forcible reasons in the particular case, but, on the contrary, upon general experience gained by much observation at the bedside, which may be expressed by saying *that we cannot be too cautious with these diagnoses.* Any one who has observed many of these cases knows how often and how readily mistakes and diagnostic errors arise, even when everything has been carefully investigated in logical sequence. My method of reasoning in such a case is the following:

The diagnosis obtained by direct analysis, it is true, primarily and immediately leads to the presumption, just expressed, of an intestinal constriction or volvulus which is the result of peritoneal adhesions arising from the genitalia. But—as has been taught us by quite analogous cases—something else might be present, and it is advisable to keep these other possibilities always in mind. What conditions must be considered here? Basis of support is given by two factors: The very insidious onset, and the seat of the stenosis in the lower portion of the small intestine. Both these facts coincide with the assumption of cicatricial stricture or neoplasm. In fact long experience has taught me to formulate the following law: When there is an unquestioned stenosis of the ileum, and absolutely no point by which we may recognize its anatomical nature, we should primarily consider cicatricial stricture following tuberculous ulcers of the ileum, even although no other evidence of tuberculosis can be determined. I have seen conditions exactly as in this case; i. e., stenosis of the small intestine in women in whom peritoneal bands originating from the genitalia were palpable, yet nevertheless, at the operation or autopsy, not these but cicatricial strictures after healed tuberculous ulcers of the ileum were shown to be the cause of the stenosis; or, a small neoplasm, either carcinomatous or tuberculous, not palpable, situated in the lowest portion of the ileum or at the ileo-cecal valve, might also be considered.

From this framework the diagnosis must be evolved; we cannot express ourselves more definitely or more carefully. *Our diagnosis, therefore, in this case is as follows: Peritoneal constriction or volvulus, or a tuberculous cicatricial stricture or neoplasm (carcinoma, tuberculoma).*[1]

PROGNOSIS

The prognosis of constriction of the intestine is always serious although not always equally so. The gravity is determined less by the seat than by the nature of the obstruction. In some forms, although naturally very rarely, spontaneous recovery may result; a benign polypoid neoplasm, even an invaginated portion of the intestine, may be thrown off, an exudate which causes pressure may be resolved, a uterus in abnormal position may be replaced, and thus the cause of the stenosis is removed. But these are all exceptions.

Without treatment stenosis almost always causes serious consequences. If no complications occur, the course of a benign stenosis is as follows:

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[1] I here include the following:

Laparotomy revealed the existence of pelvic peritoneal bands; but these were chiefly found in the large intestine, and there was no causal connection with the stenosis of the ileum. In the ileum not one but two stenoses were found, removed about 6 cm. from each other. A portion of the intestine about 13 cm. in length was excised; examination revealed two ring-shaped, coarse, fibrinous, cicatricial strictures. The mesentery belonging to this coil of ileum contained glands which were hard and the size of a hazelnut; microscopic examination revealed tubercular disease. Accordingly the intestinal strictures, also, must be looked upon as originating from tuberculous ulcers.

For a long time the compensatory hypertrophy of the musculature overcomes resistance, making the propulsion of the intestinal contents possible. Gradually, however, insufficiency of the musculature appears; it becomes paretic; fecal stasis follows, and finally, all the consequences which characterize intestinal occlusion—operation or death. In other cases the changes in the mucous membrane above the stenosis determine the further course. Diffuse peritonitis or sacculated fecal abscesses may result from decubital ulcers, also severe septic conditions, or perforation with its fatal issue. In carcinomatous stricture the process is hastened by the malignant nature of the growth.

Here and there intermediate conditions may add to the constriction and lead to sudden and complete occlusion. Thus, solid foreign bodies (fruit-stones, asparagus) or even a coarse particle of feces may completely occlude the narrow opening of the intestinal lumen. Or the heavy intestinal coil above the stenosis which is filled with feces may descend, causing torsion of the axis and volvulus. If the antecedents are unknown, or have been disregarded, cases of this kind may occasionally simulate acute intestinal occlusion. Sometimes the true condition may be suspected if active peristalsis and rigid stiffening arise, for these permit the conclusion of hypertrophy of the intestinal musculature; therefore, of an obstruction which has existed for a long time.

TREATMENT

In conclusion we must discuss the treatment.

This may be instituted with two objects: To limit the disease to a *symptomatic course,* or to attempt the *radical removal of the obstruction.* We shall begin with the latter.

Except in very isolated cases the removal of the stenotic obstruction is possible only by surgical measures. Fecal masses in the interior of the intestine narrow it but do not cause total occlusion; an inflammatory exudate capable of resorption gives rise to external pressure; these belong to the greatest rarities. In such instances treatment must be instituted in accordance with the well-known laws which apply in the concrete case. In others only the knife can aid us; it is superfluous to explain this in detail, for a glance at the previously described anatomical causes of constriction proves this convincingly. The question is only this: When shall we operate? The answer to this must be: As soon as the diagnosis of constriction has been made with certainty. For, if left to itself, the condition can never ameliorate but steadily becomes more serious. In the case of carcinoma, operative interference as early as possible is, in fact, our duty, for, apart from the consequences due to the stenosis, the danger of metastasis from the prolonged existence of the neoplasm constantly increases, and the chances of a successful operation constantly decrease. In cicatricial strictures and in constricting indurations we are not influenced by this latter consideration, but even a speedy operation is indicated, as a spontaneous recovery is not to be expected under any circumstances. Nevertheless, here we may more reasonably temporize a little, and only insist upon the operation when the clinical conditions of the pathologic picture (unbearable pain, transitory intestinal occlusion) make it necessary.

Symptomatic treatment must be based upon three different points of view: The period of time between the individual attacks of colic, the paroxysms themselves, and the appearance of complete intestinal occlusion.

The first object is attained by regulating the diet and evacuating the bowels. Large quantities of food should never be taken at one time. Its quantity must be definitely arranged; everything is to be absolutely avoided that is unsuitable in volume to the digestive powers, or which might even itself produce an obstruction, such as rye-bread, slices of potato, fibrous and tendinous meat, above all, most vegetables and fruit with their skins and seeds. It is best only to give pappy, finely divided, or finely chopped food; in the more marked grades of stenosis only fluid or semi-solid food.

It is clear that a regular daily evacuation of the bowels must be carefully brought about. According to the seat of the stenosis serviceable remedies are irrigations (of water, oil, or soap and water) or purgatives administered internally, among which the neutral salts and purgative waters, preparations of senna or rhamnus and colocynth are preferable.

The attacks of colic necessitate the complete withdrawal of food and the employment of purgatives; if active peristaltic movements and intestinal stiffening which show activity above the stenosis are present, irrigations but no eccoprotica by the mouth are indicated. Where the pain is unbearable, warm compresses to the abdomen, and occasionally even an opiate must be resorted to.

A further indication may be the appearance of complete occlusion. It is of the greatest importance that we understand clearly in the individual case how this serious result has arisen, as very different measures may be necessary in the relief of this condition.

If the stenosed area is still permeable, and only accidentally occluded by compact intestinal contents, irrigations and salines which reduce the intestinal contents to a fluid may relieve this condition.

If paresis of the musculature above the constriction is the cause of the so-called symptoms of ileus, to these evacuants we add the employment of electricity and massage, also gastric lavage, and a trial of the atropin treatment which will be described more in detail in the article upon complete intestinal occlusion. These non-surgical measures must be carried out with a clear conception of the anatomical conditions and functional disturbances. Their employment is limited to a very brief period. As soon as stasis of the intestinal contents assumes a threatening aspect, particularly as soon as the cardiac action begins to weaken, operative interference affords the only hope of saving life. This should never be long postponed, even although strangulation of the intestine have not supervened, which, in acute occlusion, causes very fulminant symptoms; a possible cardiac insufficiency should always be taken into consideration. What the operative process would be in a concrete case cannot be here described.

II. INTESTINAL OCCLUSION

The second history which I shall relate refers to an acute invagination of a portion of the intestine into an adjacent part. This process led to the complete closure of the intestinal lumen, and produced a symptom-complex which is usually grouped under the designation "ILEUS." As this symptom-complex depends not only upon mechanical closure of the intestine but also, in spite of complete permeability, upon paralysis of the musculature of the intestine, in the former case we speak of "mechanical," in the latter of "paralytic" or "dynamic" ileus. The clinico-symptomatologic limits of this term are by no means exact or sharply defined. It appears to me that occasionally there is danger in the use of this elastic term, inasmuch as by its use in a concrete case the impression may be given that the diagnosis has been clearly established. For this reason, I think it wise to reject the term ileus, although it is undoubtedly useful on account of its brevity. In its place, however, in every case where possible, not only a functional but an anatomical diagnosis should be made.

PATHOLOGY

The pathology of intestinal occlusion is much more complicated than that of intestinal stenosis. In a single concrete case it is impossible to analyze all of its features; therefore, in describing this condition I shall pursue a method different from that used in presenting stenosis.

Let us then begin upon the firm foundation of the *anatomical changes.*

In occlusion the permeability of the intestine is completely arrested. Neither compact, nor fluid, nor gaseous contents pass downward. As in stenosis a marked contrast exists between the intestinal coils situated above and those below the occlusion; the picture, however, is not uniform, but varies according to the causal relations of the process.

In those cases in which a slowly progressive stenosis gradually leads to complete occlusion, the same anatomical condition is found which I sketched in describing stenosis; often there is decided thickening of the intestinal wall, with catarrhal and ulcerative implication of the mucous membrane above the area of occlusion.

On the other hand, in acute occlusion which occurs suddenly in an intestine previously healthy, the distention of the upper intestinal coils is occasionally enormous, but the wall is not thickened by muscular hypertrophy; on the contrary, according to the nature of the anatomical process resembling formed feces may be discharged from the mouth, but more minute investigation reveals that they are composed of coagulated milk the surface of which has been stained yellow by bile. True, formed feces may be vomited in consequence of the rare occurrence of a communication between the stomach and the colon; but this phenomenon cannot be considered as a parallel with fecal vomiting in intestinal occlusion.

The opinion previously maintained that fecal vomiting denotes positively that the seat of the occlusion is in the colon, or, at least, in the lower portion of the ileum, has for some time been recognized as erroneous. We now know that this symptom may arise even in occlusion of the upper part of the jejunum. The explanation is simple. The disagreeable odor caused by the decomposition of albumin bodies, and which lends to the intestinal contents their feculent character, occurs also under the influence of definite varieties of bacteria. A prolonged action is necessary for this, such as occurs normally in the large intestine, but not, however, in the contents which rapidly pass through the small intestine. If, on account of occlusion, the contents are retained for a long time in the small intestine, here also they may denote a feculent composition. *Vice versa,* this explains how fecal vomiting may be absent even with absolute occlusion of the intestinal passage: namely, when death occurs very early, or when severe vomiting does not permit prolonged stagnation.

As to the manner in which fecal vomiting occurs, there is unanimity of opinion. In the first place, it is not caused by a *motus antiperistalticus.* It is true there is actual antiperistalsis, but the requirements for its development under the conditions here operative are not present. A detailed exposition of this interesting question is at this point impossible. Usually the explanation given by Haguenot-van Swieten is accepted. If the intestine is occluded, more or less rapidly the portion above the occluded area is filled with gas and fluids furnished by the gastric contents and the secretions which enter the intestine and attempt to pass onward. When the stasis reaches a point immediately above the obstruction, formed by the distended intestinal wall, the retained mass is gradually forced upward in the direction of the stomach. Vomiting now occurs which may be due to any cause. During this act the diaphragm and abdominal muscles contract, the area of the abdominal cavity diminishes, and pressure is exerted upon the intestine and its contents. The volume of the fluid contents cannot be propelled downward toward the anus on account of the insurmountable obstruction, nor can it be pressed upward, and thus, in a retrogressive manner, it is forced toward the stomach, and fecal vomiting results. It is readily perceived that, analogous to the act of vomiting, the contractions of the intestine, moving in a normal direction, must mechanically affect the contents in its lumen, thus causing a regurgitation of the contents. That the act of vomiting as such, and not antiperistalsis, is the cause of the expulsion of the intestinal contents is borne out by an experience of my own that in an enterostenosis of the sigmoid flexure the entire colon and the small intestine up to the jejunum were filled with thin feculent contents, yet fecal vomiting did not occur. The patient did not vomit during the entire course of the disease and retching even was not present.

Meteorism occurs, but by no means in the same form and to the same extent in all cases. This depends upon the etiologico-anatomical form of the intestinal occlusion. It is true that the passage of flatus ceases entirely, but the more important resorption of gas from the intestine into the blood may continue as long as a simple stenosis of its lumen without an alteration of its wall exists, and in case the formation of gas due to the accidental presence of abnormal quantities of fermentative material does not preponderate over the absorption of gas. Under these circumstances meteorism will be but moderate. This is more frequently observed in occlusion which develops from stenosis of the intestine; quite as frequently also in the case of internal occlusion of the intestine (*obturatio intestinalis*) by foreign bodies, gall-stones, intestinal stones and fecal masses; and also in cases of external compression. On the other hand, in the forms of acute occlusion which run their course with strangulation of the intestinal wall and of the mesentery, marked degrees of meteorism may be observed. Here the areas nearest the occlusion show the greatest distention, and from the peculiar formation and situation of the inflated coils important data not only for the localization of the seat of the obstruction but occasionally even indicative of its anatomical nature may be obtained.

Finally, as a direct symptom of occlusion, the *increased excretion in the urine of indican and other combined ethereal sulphates* must be mentioned, of which indicanuria is the most important in practice because of the readiness with which it may be chemically determined. In occlusion of the small intestine there is a large, and occasionally enormous, increase of indican in the urine; in occlusion of the colon only when this lasts a long time, and when there is stagnation of its contents above the ileo-cecal valve. In the use of this symptom embarrassment has arisen from the fact that under some conditions which must be here considered, very frequently, in addition to the occlusion and also as produced by it, a diffuse peritonitis which runs its course with decided indicanuria is superadded.

All of the symptoms which have as yet been described are the direct or indirect consequence of the arrested impermeability of the intestinal lumen, and they appear when the cause of the impermeability in its nature has this effect only, and does not, at the same time, cause strangulation. Only in isolated cases, especially in acute obstruction by large gall-stones, do other symptoms appear which resemble the severe clinical picture of strangulation.

We shall now turn to these.

It must first be remarked that the *clinical picture in which symptoms of occlusion and strangulation appear simultaneously* is, without excepmarkedly edematous and thickened, and the contents of the sigmoid flexure consist of blood and feces. In the other forms of acute occlusion which accompany strangulation, namely, internal incarceration and invagination, local meteorism either does not occur at all or scarcely develops to such an extent as to be of clinico-diagnostic importance.

Although it has been already mentioned, the importance of the subject leads me to reiterate that in acute occlusion, if this attack an intestine previously permeable, peristalsis that is distinctly visible and tetanic intestinal stiffening only occur exceptionally, and never to the extent seen in occlusion which follows a slowly growing stenosis. The reasons for this are obvious: In the first place, there is no muscular hypertrophy of the intestinal wall in acute occlusion, and the paralysis of the musculature which occurs develops with particular rapidity in the doubly occluded and strangulated intestinal areas in which local meteorism has developed; paralyzed muscles, of course, do not contract.

The extremely rare occurrence of *diarrhea* in acute occlusion (in invagination, incarceration), is deserving of brief mention, since, at first, it may lead to gross errors in diagnosis. This is explained by the hypothetic assumption of a hypersecretion of fluid from the intestinal wall in consequence of paralysis of the mesenteric nerves.

Besides this group of symptoms, other symptoms on the part of the circulatory apparatus are noted; these dominate the situation, and impress their stamp upon the affection, the clinical condition closely resembling "collapse" from other causes. The extent of these phenomena may vary in individual cases; as a rule, however, they are decidedly marked.

In regard to their occurrence two views are maintained at the present time: A nervous reflex theory, and an intoxication theory. In my opinion the latter is operative only for certain rare symptoms and cases. On the other hand, the nervous reflex theory readily explains the majority of symptoms which are clinically common and important, and which usually appear at the onset of the affection.

I cannot here analyze the reflex theory in detail, but will only remark that in accordance with well-known physiological facts the severe clinical picture is caused by decided irritation of the sensory nerves of the intestine and of the peritoneum; that is, of the mesentery, which results with the acute onset of volvulus, incarceration, invagination, obstruction from gall-stones, etc. Occlusion thereby leads to an alteration of cardiac activit and of the entire distribution of the blood, this being brought about b the reflex influence of the cardiac vagus and of the splanchnic nerve. I fact, the majority of the symptoms may be thus explained, particularl those above described and also some rare ones; as, for example, the albu minuria and cylindruria which are occasionally observed in incarcerate hernia.

The idea of the "intoxication theory" is this, that in the closed i testinal passage the arrested products of bacterial proteid decomposition which had formed in large amounts (phenol, indol, skatol, the aromatic oxyacids) are absorbed by the blood, and thus exert their toxic action. A modification of the intoxication theory, designated as "septic collapse," assumes that the intestinal microbes reach the peritoneum through the intestinal wall, which is damaged by the strangulation, and thence, even without causing peritonitis, infect the entire organism. The following may be stated in criticism of these views: It is possible that a few very rare symptoms of strangulation shock may be referred to intoxication of this kind, and among these delirium or coma, rise of temperature without peritonitis, and the so-called "incarceration typhoid." But the facts have by no means been determined with certainty, and against its general acceptance very important clinical objections may be raised; for instance, that the severest picture, the most profound collapse, may develop in from six to twenty-four hours after the occlusion; that, as a rule, the intoxication is in proportion to the suddenness of the incarceration and the severity of the initial pain; that in occlusion which develops gradually it may be entirely absent.

The course of the disease is not always the same; it varies according to the anatomical foundation, the cause of the occlusion, and particularly according to whether simple occlusion or accompanying strangulation exists.

In occlusion with strangulation occurring suddenly, death may follow in a few hours (eight) with all the symptoms of collapse. At other times peritonitis is added and brings about the lethal termination. Its development in these forms is favored by the fact that in the area of the strangulated region there is a histological alteration in the intestinal wall which makes it possible for microbes to penetrate into the peritoneal cavity. To this already alarming picture more circumscribed or distributed spontaneous painfulness upon pressure is added, according to the distribution of the peritonitis. This peritonitis may, although rarely, develop in pure occlusion. On the other hand, if no relief is brought about, death usually occurs by gradual exhaustion in consequence of the absence of nourishment and of continuous vomiting.

What are the conditions which lead to complete intestinal occlusion?

I have already mentioned quite a number of the causes under intestinal stenosis (which see). All, or nearly all, of the processes there enumerated are capable, either directly or indirectly, of causing complete occlusion of the intestinal lumen. They furnish the cases in which occlusion forms, as a rule, by gradual stages from the constantly advancing stenosis, and in which the clinical picture is composed of the symptoms of pure occlusion. Exceptionally, and under special circumstances, the stenosis suddenly and rapidly increases to entire impermeability; as when a coarse foreign body entirely occludes the narrow passage, or the dilated p the intestine above the stenosis, which is filled with contents, des leads to volvulus and the like.

A *second series* of cases is furnished by complete occlusion of th tinal lumen by any large impacted masses there present (obstru the intestine). Of this variety are:

(a) Enormous fecal masses which, in addition to their dire pating effect, produce a paralytic weakness of the musculature intestine;

(b) Intestinal calculi;

(c) Gall-stones;

(d) Foreign bodies.

In this series of cases the clinical picture exhibits exclusively th toms of occlusion. Only in obstruction due to gall-stones are symp collapse occasionally observed which, as stated previously, are prod the severe sensory irritation of the stone upon the intestinal wall.

A *third series* of cases is formed by processes which not only oc clusion but, at the same time, produce strangulation of the intestin is, of the mesentery:

1. Volvulus;
2. Noose formation—which, at this time, is usually included und designation volvulus;
3. Hernia, internal incarceration;
4. Invagination, intussusception.

These conditions almost invariably occur acutely, and are accomp by the distressing symptoms of the clinical picture last mentioned.

In conclusion, an affection must be mentioned which mechanicall not the remotest connection with occlusion of the intestinal lumen whose physiologic termination is nevertheless the same.

This is *paralysis of the intestine*. Here, the lumen is wide open passage mechanically unhindered; nevertheless, its function has because motor paralysis in a large or small portion of the intestine m the propulsion of the contents through this area impossible.

We require no explicit description to make it clear that, besides t conditions which in combination form the clinical picture of occlu or of occlusion with strangulation added, each individual one may duce in the clinical picture its own distinctive symptoms. In this art which is merely a comprehensive review of intestinal occlusion, it is im sible to enter more minutely into the subject. Each of the various co tions would require special description.

DIAGNOSIS

lthough the analysis of the symptoms which make up the clinical pic- of occlusion is simple, in reality it may be extraordinarily difficult to the *diagnosis at the bedside.* In fact, when the individual points onsidered, and occasionally even in deciding the fundamental ques- this proves a most difficult task for the physician. The most expe- ed and the most careful investigator is liable to err, and yet, in this e affection, not only is certainty but, especially, rapidity of diagnosis e utmost importance in the treatment.

n each individual case the diagnosis must be based upon the answers ree distinct questions:

1. Is intestinal occlusion present?
2. In what portion of the intestine does it exist?
3. What is the anatomical foundation of the condition?

In regard to the first question: *Is intestinal occlusion present?* Errors iagnosis may be made in two ways. Intestinal occlusion is assumed use the symptom-complex is more or less completely developed; yet, ithstanding, this condition is not present, but the symptoms are due nother affection. Or the symptoms, which but partially indicate the ition, divert the attention of the physician into another channel, and actually existing occlusion is overlooked.

The diagnosis is based mainly upon the local intestinal symptoms: Ab- te impermeability for gas as well as feces, meteorism, vomiting, fecal iting.

That the first three symptoms, taken individually, furnish but limited f need not be dwelt upon. Even fecal vomiting is of no pathogno- ic diagnostic value. Apart from its possible appearance in hysteria, from its actual but very rare occurrence in fistulas between the colon stomach, it may also appear—as well as absolute constipation, meteor- , and vomiting—in intestinal occlusion from paralysis of the intestine from peritonitis (in the latter instance also it is pathogenetically due ntestinal paralysis). Under the following circumstances, the physician t consider the possibility of pure intestinal paralysis without mechan- occlusion: When fecal vomiting appears after some dull trauma has ted the abdomen; when it persists in incarcerated hernia after the hanical obstruction has been removed by the knife or other means; in ral, when it appears after surgical operation upon the intestine itself n its immediate vicinity; or when it has developed in disease of the cles, in the inguinal region, in the inflammation of hemorrhoids, in l colic, in gall-stone colic, in recent acute appendicitis and perityph- . To this must be added the possibility of the appearance of intes- l paralysis in embolism of the mesenteric artery, in habitual constipa- , in over-distention from excessive meteorism and, perhaps, also in consequence of bacterial peritoneal infection without peritonitis. all these circumstances fecal vomiting and the entire symptom- *may* be caused by pure intestinal paralysis, and the physician mu bear this possibility in mind, and should never forget that in the case actual mechanical occlusion may also be present.

The most frequent and the greatest perplexity is caused by the qu Occlusion or peritonitis, or their combination? Its vital importa strains me to dwell somewhat more explicitly upon the individu which may aid us in the differential diagnosis.

The pain is of great significance. Severe pain occurs spontane acute peritonitis as well as in acute occlusion. If to this, however, well-developed tenderness of the entire abdomen upon palpation added, if the patient lies very quiet and carefully avoids voluntar ment, this is more in favor of peritonitis. Naturally, the latter co may very rapidly develop in occlusion, but scarcely sooner than forty-eight, or, at the earliest, twenty-four hours. The possib examination within the first or the second day may, therefore, important data to clear the diagnosis. Of course absence of pain pressure is by no means absolute proof of occlusion and disproof of tonitis, for, on the one hand, even in acute and purulent peritonitis taneous pain as well as pain upon pressure may be only of minimal and, on the other hand, the rapidly developing meteorism in volvulu in internal incarceration may occasionally run its course with quite sensitiveness to pressure; nevertheless, decided painfulness upon pre more strongly indicates peritonitis.

Sometimes in acute cases the degree of meteorism will establis diagnosis. If, at the onset, it develops decidedly as local meteoris it is possible to detect circumscribed, inflated, extremely tense p of the intestine by simultaneous percussion and auscultation, we ma sume volvulus or an internal incarceration. Diffuse meteorism, on the trary, may be observed in occlusion as well as in peritonitis, and for reason cannot be utilized in a differentio-diagnostic respect; the enor balloon-like inflation makes an accurate examination impossible.

The significance of the intestinal movements in such conditions been several times described. I believe it to be certain that positive, de peristalsis and, particularly, paroxysmal intestinal stiffening always an obstruction in the passage. Inversely, if it has been determined careful examination by means of inspection, palpation, and ausculta that there is absolutely no sign of intestinal movement, this points great likelihood to peritonitis with intestinal paralysis. But the oc rence of intestinal movement is, unfortunately, not absolute and pos proof. For complete stoppage may also occur without peritonitis in co quence of enormous over-distention, and, inversely, it may continue after the development of peritonitis.

ie presence of a fluid exudate is in favor of peritonitis, but here it is
ary for us to be cautious; since, on the one hand, in internal incar-
on, particularly in volvulus in consequence of mesenteric venous
, such a profuse hemorrhagic transudation may occur in the perito-
cavity that a peritoneal effusion is simulated; on the other hand, the
may be actually present, but, because masked by peculiar physical
tions, may not be recognized.

ever points to peritonitis; but the absence of the same does not ex-
peritonitis, as any form of this affection may sometimes run its
se without fever and even with subnormal temperatures.

ccasionally the presence of indicanuria may be of diagnostic impor-
e, naturally only under very definite conditions. The positive proof of
ased indicanuria coincides with the existence of a diffused peritonitis
ell as an occlusion of the small intestine. However, its absence is
nst the presence of either of these processes, and the same is true of
cclusion of the large intestine, for example, volvulus of the sigmoid
re.

Naturally other factors may be utilized in the diagnostic consideration
he concrete case. For example, the presence of florid ulcerative pro-
es in the stomach or intestine is an indication of peritonitis. Care-
analysis will very frequently reveal the true situation, but there are
s in which the diagnosis, either of peritonitis or intestinal occlusion,
be impossible.

If the presence of intestinal occlusion has been determined the question
t arises: *In what portion of the intestine does it exist?* To answer this
xtremely difficult. As a positive aid it is well first to determine clearly
ther the occlusion has developed from stenosis or whether its onset was
te, that is, simultaneously with symptoms of strangulation. In the
ner case its localization is readily decided, and as guides, the factors
ch I have mentioned in the article upon intestinal stenosis, to which
ere refer, may be utilized.

If we are dealing with acute occlusion, that is, with strangulation, the
st minute manual exploration of the hernial rings, as well as of the rec-
and vagina, is necessary. Occasionally, by this means, important data
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consequence of bacterial peritoneal infection without peritonitis. Under all these circumstances fecal vomiting and the entire symptom-complex *may* be caused by pure intestinal paralysis, and the physician must always bear this possibility in mind, and should never forget that in the concrete case actual mechanical occlusion may also be present.

The most frequent and the greatest perplexity is caused by the question: Occlusion or peritonitis, or their combination? Its vital importance constrains me to dwell somewhat more explicitly upon the individual points which may aid us in the differential diagnosis.

The pain is of great significance. Severe pain occurs spontaneously in acute peritonitis as well as in acute occlusion. If to this, however, diffused, well-developed tenderness of the entire abdomen upon palpation is soon added, if the patient lies very quiet and carefully avoids voluntary movement, this is more in favor of peritonitis. Naturally, the latter condition may very rapidly develop in occlusion, but scarcely sooner than after forty-eight, or, at the earliest, twenty-four hours. The possibility of examination within the first or the second day may, therefore, give us important data to clear the diagnosis. Of course absence of pain upon pressure is by no means absolute proof of occlusion and disproof of peritonitis, for, on the one hand, even in acute and purulent peritonitis spontaneous pain as well as pain upon pressure may be only of minimal degree, and, on the other hand, the rapidly developing meteorism in volvulus and in internal incarceration may occasionally run its course with quite marked sensitiveness to pressure; nevertheless, decided painfulness upon pressure more strongly indicates peritonitis.

Sometimes in acute cases the degree of meteorism will establish the diagnosis. If, at the onset, it develops decidedly as local meteorism, if it is possible to detect circumscribed, inflated, extremely tense portions of the intestine by simultaneous percussion and auscultation, we may assume volvulus or an internal incarceration. Diffuse meteorism, on the contrary, may be observed in occlusion as well as in peritonitis, and for this reason cannot be utilized in a differentio-diagnostic respect; the enormous balloon-like inflation makes an accurate examination impossible.

The significance of the intestinal movements in such conditions has been several times described. I believe it to be certain that positive, decided peristalsis and, particularly, paroxysmal intestinal stiffening always shows an obstruction in the passage. Inversely, if it has been determined after careful examination by means of inspection, palpation, and auscultation that there is absolutely no sign of intestinal movement, this points with great likelihood to peritonitis with intestinal paralysis. But the occurrence of intestinal movement is, unfortunately, not absolute and positive proof. For complete stoppage may also occur without peritonitis in consequence of enormous over-distention, and, inversely, it may continue even after the development of peritonitis.

The presence of a fluid exudate is in favor of peritonitis, but here it is necessary for us to be cautious; since, on the one hand, in internal incarceration, particularly in volvulus in consequence of mesenteric venous stasis, such a profuse hemorrhagic transudation may occur in the peritoneal cavity that a peritoneal effusion is simulated; on the other hand, the latter may be actually present, but, because masked by peculiar physical conditions, may not be recognized.

Fever points to peritonitis; but the absence of the same does not exclude peritonitis, as any form of this affection may sometimes run its course without fever and even with subnormal temperatures.

Occasionally the presence of indicanuria may be of diagnostic importance, naturally only under very definite conditions. The positive proof of increased indicanuria coincides with the existence of a diffused peritonitis as well as an occlusion of the small intestine. However, its absence is against the presence of either of these processes, and the same is true of an occlusion of the large intestine, for example, volvulus of the sigmoid flexure.

Naturally other factors may be utilized in the diagnostic consideration of the concrete case. For example, the presence of florid ulcerative processes in the stomach or intestine is an indication of peritonitis. Careful analysis will very frequently reveal the true situation, but there are cases in which the diagnosis, either of peritonitis or intestinal occlusion, may be impossible.

If the presence of intestinal occlusion has been determined the question next arises: *In what portion of the intestine does it exist?* To answer this is extremely difficult. As a positive aid it is well first to determine clearly whether the occlusion has developed from stenosis or whether its onset was acute, that is, simultaneously with symptoms of strangulation. In the former case its localization is readily decided, and as guides, the factors which I have mentioned in the article upon intestinal stenosis, to which I here refer, may be utilized.

If we are dealing with acute occlusion, that is, with strangulation, the most minute manual exploration of the hernial rings, as well as of the rectum and vagina, is necessary. Occasionally, by this means, important data are revealed, particularly in incarcerated external hernias.

Irrigation of the rectum, which is much practised in order to determine the seat of the occlusion from the amount of fluid which may be injected, is sometimes impracticable on account of the condition of the patient, and even when possible the result is frequently unreliable. A deep-seated obstruction may be determined only when, after repeated irrigations, but little fluid can be forced into the intestine, and this invariably returns rapidly. But we must not forget that the rectum with its ampulla may take up from one to one and a half liters of fluid. Sounding of the rectum is also unreliable.

conditions incline us to the assumption of volvulus, of incarceration, or of invagination, other points of support must be present to make possible the differentiation of these processes.

Pain is here of the less importance, because it is common, almost invariable, and usually the initial symptom in these conditions. Perhaps its primary seat may indicate the anatomical diagnosis: If extending from the left hypogastrium to the sigmoid flexure, volvulus; if in the right hypogastrium, to an invagination of the ileum and cecum; if in the middle and upper abdominal region, to an internal incarceration or volvulus of the small intestine.

Even less significant is vomiting.

Absolute constipation is the rule in internal incarceration, in volvulus, and in obstruction from gall-stones, sometimes too in invagination, but here diarrhea occasionally alternates with constipation. Hemorrhagic discharges are most frequently observed in invagination, rarely in volvulus of the sigmoid flexure, very exceptionally in internal incarceration, and not at all in obstruction due to gall-stones. The same laws exist for tenesmus as in regard to hemorrhagic discharges.

Collapse suddenly appearing may occur in all forms of occlusion, most markedly in internal incarceration, next in volvulus, and to a less degree (even entirely absent) in invagination and obstruction.

Better grounds for the diagnosis may be furnished by the local findings in the abdomen, if it be possible to obtain these. Above all, acute local meteorism may lead to definite conclusions. If we can be sure that the tensely inflated, elastic, motionless, intestinal coil rises from the left lower side of the abdomen, this is in favor of volvulus of the sigmoid flexure. Invagination may be determined with certainty if a tumor is present which possesses the characteristic properties of an invagination tumor; namely, that it is usually soft but periodically becomes hardened by rigid contractions of the wall.

TREATMENT

Among the most difficult and responsible duties of the physician is the treatment of intestinal occlusion. Upon his decided opinion, based [illegible] upon his definite conclusions, naturally, the life of the patient often depends. While the action in the case of occlusion developing from chronic causes [illegible] [illegible] to immediate interference, the situation in acute occlusion often necessitates most urgent measures; within a few hours the most far-reaching decisions must sometimes be made, and this often, too, when we are unable to recognize the seat and the nature of the occlusion, and occasionally are even in doubt as to whether an occlusion [illegible] is present. Consider [illegible] features—features [illegible] the patient, collapsed and vomiting, is found to have complete intestinal obstruction, his abdomen distended like a drum, and all diagnostic points which aid in the determination of the nature of the affection are absent! Nevertheless, the physician must act. The patient, unless already in the terminal agony, must not be left to his fate. What is to be done?

Some authors absolutely deny the occurrence of a spontaneous cure, that is, without surgical interference, in an internal incarceration, a volvulus, or kinking. Others, upon a basis of actual experience, believe this to be possible in favorable anatomical conditions and in the earlier periods of the affection, although exceedingly rare. The first contingency, however, absolutely requires operative interference; the latter permits, within definite limits and even here in a certain period of time, non-surgical treatment. Naturally, the latter does not and cannot decide the treatment, and this statement is only made to show how a very surprising course may be occasionally explained.

In regard to trêatment, we must differentiate between simple occlusion and occlusion with strangulation.

In occlusion with strangulation it is the rule to operate as soon as the symptoms of the latter become at all marked. If the diagnosis of the seat and the nature of the disease can be made, exploratory laparotomy and removal of the obstruction is in place. The chances of operation are best if it be done within the first forty-eight hours after the appearance of the symptoms of occlusion: 75 per cent. recover; after the third day, the percentage of recoveries falls rapidly to 35 to 45 per cent. It remains on this plane even when the operation is done only in the course of the second week.

If the physician recognizes that the intestinal occlusion is either internal incarceration or volvulus, no time is to be lost by attempting any other method of treatment. For the psychical effect upon very anxious patients and to relieve the minds of friends and relatives during the time when preparations for the operation are being made, at most only a brief trial of non-surgical measures is allowable.

It appears that twists, if not of marked extent, may occasionally recover under non-surgical treatment. This may also be resorted to in invagination, but in these two conditions operation should be attempted early, provided non-surgical measures do not rapidly bring relief.

If gall-stones cause intestinal occlusion, experience has shown that the outlook for a spontaneous recovery or by internal remedies is not unfavorable. In this case, we should decide upon laparotomy only when the symptoms indicate direct danger to life. The gauge for this is furnished by the cardiac activity; we shall revert to this later.

What has been said of gall-stones is also true of large and round foreign bodies. When sharp or angular substances cause obstruction purgatives must be avoided, and in their place soft food should be taken and potatoes, rice, oatmeal grits, which form a mass safely enclosing the foreign bodies.

I shall now return to the question of operation.

Experience teaches that the *chances of operation* not only depend upon its early performance, but also increase with the recognition of the nature and location of the obstruction, as this makes possible a direct attack at its seat. If, however, the diagnosis is obscure, and only peritonitis and intestinal paralysis are excluded, what is to be done? The answer must certainly be: Operate at once, provided the operation in itself would be a harmless affair. This is, however, not the case if symptoms of strangulation are at all marked. Although the modern surgeon may, with some degree of certainty, exclude septic infection in peritonitis, other dangers threaten, particularly in these laparotomies, and especially if the seat of the disease must first be searched for (intestinal paralysis, reflex influence upon the nervous system and upon the circulatory apparatus). Here, that is, with an uncertain diagnosis, two kinds of cases must be differentiated. If the course is very acute, if there are severe symptoms of shock, probably only the operation can save life. Without this almost all patients perish, and one saved by this means may be looked upon as pure gain. If, however, the course is more moderate, the entire picture not too severe, so-called internal treatment may be resorted to with a definite view and purpose which shall be later explained.

In attempting this therapy, the physician must carefully observe the *cardiac activity,* as shown by the quality of *the pulse.* In these cases the condition of the pulse will correctly indicate the best time in which to perform the operation. If the pulse begins to increase in frequency, the tension to sink but a little, it is time to operate, for somewhat later this may become impossible. The weakened cardiac activity is no longer sufficient to meet the requirements of an operation. In such cases it is much better to operate several days before this critical cardiac change than to wait but a few hours after it has taken place.

If the symptoms of occlusion exclusively appear in the clinical picture, while those of strangulation are absent, often an operation alone can save the life, but in the less fulminant course of the affection there is time to try non-surgical measures. In quite a number of cases these may entirely relieve the threatening situation.

There has been much discussion of late as to the cases of intestinal occlusion in which a resort to non-operative treatment is at all permissible, under what circumstances, and as to the factors upon which the decision depends.

Primarily, a point of support, as I have already mentioned, is the general condition as expressed in the cardiac activity and in the condition of the pulse, while the local symptoms of occlusion (meteorism, vomiting) are of less import. But, I repeat, if the diagnosis at the onset is uncertain, and if the cardiac condition is good, relief by non-surgical measures may be attempted, for, during this time, by rapid decision, we may obtain more data in support of the local and collective diagnosis to improve the chances of operation. If by the application of these principles the possibility and justification of a brief delay is gained, the question then arises which non-surgical measures are most likely to be successful.

First, a method which is *not* to be resorted to must be pointed out. In all forms of acute, subacute, chronic, and complete intestinal occlusion *purgatives are decidedly dangerous,* and for this reason must be absolutely avoided—with the single exception, in case the obstruction is due to fecal masses. With a positive or lightly acute occlusion by incarceration, torsion, kinking, or obstruction by gall-stones and foreign bodies, as well as whenever the diagnosis is questionable, purgatives should never be employed! Only when in a concrete case obstruction due to feces is almost certain may purgatives be administered by the mouth. Mercury, formerly so much used, may be entirely dispensed with, for it is occasionally directly harmful and still more so indirectly, as valuable time is lost in its employment.

Enemata and rectal irrigations are often advised, but very little more may be expected from them than from purgatives by the mouth. If they do not flow off again they also have a harmful effect, since by their massiveness they raise the already increased intra-abdominal pressure. These facts must always be taken into consideration. *A priori,* and according to experience, enemata and large injections are useless in nearly all forms of acute occlusion; the only exception to this is formed by acute invagination which, if it have occurred in the large intestine, may, in fact, be caused to disappear by forced massive enemata or by the introduction of air. In one form of chronic occlusion these are almost indispensable, and combined with internal purgatives they form a most valuable therapeutic agent, namely, in obstruction from feces, immaterial whether this takes place in a free atonic intestine or above the point of stenosis. But we must be very careful in their use if there is a well-founded suspicion of ulcerative processes or fissures in the intestine.

For irrigation, water (1–5 liters) or oil (½–1 liter) is employed. The temperature of the water should be that of the air; ice-water injections are no more beneficial, and are contraindicated in all debilitated patients. Highly irritating additions, particularly of table salt (5 to 8 per cent.), are perhaps only of use in acute invagination. The value of the irrigation depends, in the main, upon its softening property, and for this purpose pure water, or soapy water, or oil is sufficient. The fluid must always be introduced with only slight force and very slowly, from one-half to one hour being required.

Lately carbonic acid enemata have been employed, of which about the same is to be said as of injections of water. The inflation of gas into the rectum, as occasionally practised, is naturally useless in obstruction due to feces; in the acute forms of occlusion the same is true as of the irrigations with water, i. e., an effect is only occasionally to be expected in acute invagination.

Massage of the abdomen, as well as the application of the faradic current in the milder degrees of the affection, may prove useful in a single form, namely, that of obstruction due to feces. On the contrary, massage as well as faradism is to be absolutely rejected in all forms of acute occlusion with strangulation, and also when the possibility of peritonitis or hernia may be assumed, which, in the case of strangulation, often begins very early. In invaginations of very recent origin, massage (with simultaneous narcosis) is said to be occasionally useful; but even here the greatest caution is necessary and, in my opinion, rectal injections and inflations are generally more successful and considerably less dangerous.

An important enrichment of our therapy in acute intestinal occlusion, *gastric lavage*, has been introduced by Kussmaul. Its value consists in this, that the evacuation of the gastric contents facilitates the regurgitation of the intestinal contents into the stomach. This causes the cessation of retching and fecal vomiting and a subjective feeling of amelioration is produced. Experience shows, however, that the employment of gastric lavage occasionally does even more than this; an absolute cure of the severest forms of complete intestinal occlusion in cases lasting from eight to nine days has been brought about by its use. This treatment should not be deferred until too late, and we should never rest content with a single lavage, but the method must be employed frequently, often being repeated every two or three hours, since the regurgitation of the contents of the small intestine into the stomach may take place with extraordinary rapidity. The same objection has been made to gastric lavage as to opium treatment, that it brings about a deceptive euphoria while the local process remains unchanged or advances. As stated, these agents occasionally have a curative effect, and, apart from this fact, the patient who suffers severely must not have such a beneficial remedy withdrawn from him even although it have but a symptomatic effect. For it is the duty of the intelligent and conscientious physician constantly to keep in mind the question of an operation, and, in spite of apparent subjective improvement, closely to observe the most decisive local symptoms (passage of feces and flatus) as well as to watch over the cardiac activity.

I must still refer to two drugs used internally. One of these is *opium and its preparations*, which may be administered either by the mouth or by subcutaneous injection in the form of morphin. There is great difference of opinion as to their use. That they may be given, or must be given, to tide the suffering patient over a few hours of misery when the situation is hopeless and operative interference is no longer possible, is generally recognized. In some cases its employment is permitted by even its most bitter opponents; for instance, if in high-graded stenosis, enormous peristalsis and intestinal stiffening make us fear early paralysis of the musculature of the intestine, and if, during this time, attempts have been made to overcome the obstruction by rectal irrigation and other measures. The dispute, on the contrary, is concerning the question whether or not opium may be given, that is, must be given at the beginning of acute occlusion. Some surgeons are decidedly opposed to its use for the following reasons: Because the treatment by opium brings about only an apparent improvement, and by masking important symptoms renders the diagnosis difficult, increases the danger of collapse, and facilitates the onset of intestinal paralysis; also because the patient, on account of apparent improvement, rejects an operation. In opposition to this the following may be said: The feeling of intense anxiety, the nausea and vomiting, the severe pains, frequently cease. If given at the right time, i. e., immediately at the onset, opium occasionally has a surprising effect upon the symptoms of initial reflex collapse. And even if this were its only effect—which is not the case, as it occasionally influences peristalsis, even, perhaps, makes possible the spontaneous improvement of the anatomical disturbance—treatment by opium should not be rejected for it brings great relief to the suffering patient. The dreaded deleterious effects may be obviated if, as I have just remarked concerning gastric lavage, the physician does not permit himself to be deceived, and continually keeps in mind the local intestinal symptoms and the cardiac condition; if, moreover, the employment of opium is instituted only upon the first or second day of the disease, and, notwithstanding its apparently good effect, the question of operation is decided according to the principles which I have indicated.

The other remedy, quite recently introduced into the treatment of "ileus," is *atropin* given subcutaneously in decided doses, 0.001 to 0.005 at a dose, and repeated if necessary. In the last few years a series of cases has been reported in which atropin was used under most desperate circumstances when for many reasons an operation was out of the question; it freed the intestinal tract, and recovery followed. It is impossible to base an opinion upon the statistics at hand. In the cases in which atropin was ineffectual, an anatomical intestinal occlusion was shown. In the single case in which I have had opportunity to observe its favorable effect, the symptoms were those of severe intestinal occlusion in consequence of obstruction due to gall-stone. I presume that the effect of atropin in intestinal occlusion may best be observed when the underlying condition is that of a spastic or paralytic obstruction.

In acute occlusion the *ingestion of food* must be absolutely suspended from the first day. When the thirst is tormenting small pellets of ice may be given by the mouth or minimal amounts of ice water may be swallowed. In case of profuse loss of fluid by vomiting, fluid may be inj into the rectum or subcutaneous infusions of normal salt solution ma employed. Where the course of the disease is prolonged, nutritive ene are to be advised.

For severe cases of well-developed intestinal paralysis the meas previously described (massage, electricity) have proven ineffectual. Fr ther experience will show whether physostigmin (physostigmin salicyl 0.0005 per dose), recently introduced by v. Noorden, will furnish any b ter results.

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